THE HUNTERIAN LECTURES

1904

ON

FRACTURES OF THE SKULL

Sir Lauder Brunton

With the Author's
compliments

June 1904

FRACTURES OF THE SKULL

THE HUNTERIAN LECTURES

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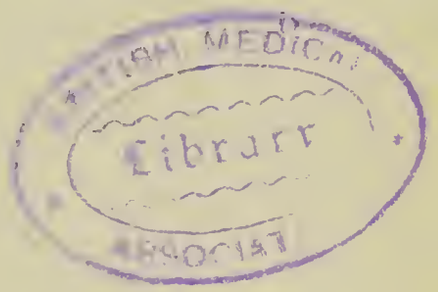

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PREFACE.

It was my original intention to withhold these Lectures from publication till some future date when further research should have enabled me to add other details to the study of this interesting subject. I have had, however, so many requests for "reprints" that I determined to follow out the suggestion, trusting that these Lectures will be found of surgical value, and that they may form another stepping-stone in the elucidation of the many obscure features in cases of head-injury. The illustrations are all from original specimens, many of which are now in the possession of the Royal College of Surgeons.

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May, 1904.

FRACTURES OF THE SKULL.

LECTURE I.

Delivered on Feb. 29th.

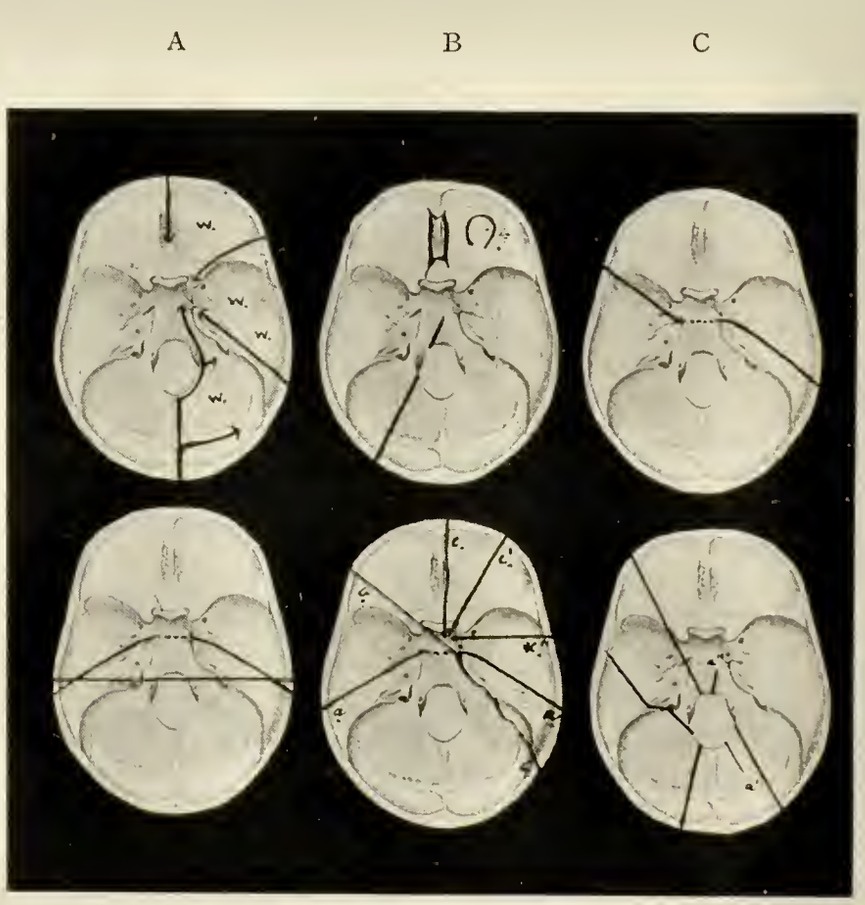
Mr. President and Gentlemen,

The subject which I have chosen for these lectures is one of exceptional interest on account of the obscurity of many of its details. In every large hospital abundant material is offered for the elucidation of many of these obscure features in cases of head injury, features which have been for many years the subject of discussion and dispute. The complete question of skull fractures and of the numerous complications, immediate and remote, which may subsequently ensue would require a much greater period of time than that which I have at my disposal and consequently I propose to discuss only certain of the more interesting points, limiting myself as far as possible to facts and theories which are either omitted or superficially treated in the majority of text-books. It is necessary first of all to mention briefly certain facts in connexion with the anatomy of both vault and base.

The vault varies in density in different cases to a remarkable extent, but in all it can be said to present areas of greater or lesser resistance, according to the density of the internal and external tables and the amount of intervening diploe. Ridges of bone, also, pass up from base to vault, ribbing and strengthening the skull, whilst the intervening areas, being weaker, offer a lesser resistance to blows. These bony buttresses pass up from (1) the crista galli; (2) the external angular frontal process; (3) the mastoid process; and (4) the occipital protuberance. As the result of a blow on the vault the forces are to a large extent transmitted to, and conducted along, these ridges towards the basic fossæ, whilst during their transmission in that direction a "deadening" of the forces is produced in several ways. 1. The convexity of the vault itself helps to distribute the forces over a large superficial area though the forces necessarily travel mainly in the direction of the applied

force. 2. The intervention of fibrous tissue or of cartilage between two or more bones assists in the "deadening" of the transmitted forces. 3. The bony ridges themselves come to an end, terminating in front at the crista galli, in the antero-lateral region converging to the anterior clinoid processes, and in the lateral region to the apex of the petrous bone. Posteriorly the forces are transmitted laterally along the lateral sinus ridges and forwards along the internal occipital crests to

FIG. 1.



For explanation of the diagrams see context.

the strengthened margin of the foramen magnum and skirting that foramen the forces pass laterally to the jugular process of the occipital bone or forwards to the dorsum ephippii and to the posterior clinoid processes (Fig. 1, A). 4. The supporting layers of the dura mater through their attachment to the above-mentioned bony ridges and prominences must necessarily receive a large share of the transmitted forces. The figure also illustrates the great tendency of numerous forces to converge towards the pituitary region, which region is practically

inclosed by the anterior and posterior clinoid processes and to each of these processes the tentorium cerebelli is attached. Now it is exceedingly common to find in the more severe basic fractures that these processes are torn away from their basic attachment. Especially is this the case with regard to the anterior processes which are frequently expanded at their apices and attenuated at the base, and in such cases no great force is needed to snap them off. A similar condition is, however, not infrequently seen when the processes are blunted and strong. The diagram also illustrates the main bony barriers along which the forces are conducted to the base and it is manifest that these ridges, with the intervening weaker areas, must play an important part in influencing the direction of a basic fracture.

The base of the skull is, again, in itself in many respects the weakest part of the skull. It is perforated by numerous foramina; it is hollowed out in places to form air sinuses and also to form a cavity for the integral parts of the auditory apparatus. One would therefore expect that basic fractures show some predilection for these weaker areas. The base also lies practically in one plane, the forces, therefore, being transmitted straight across the base and not diffused, as is the case when a blow is applied to the vault. It would consequently appear probable that a fracture passing from vault to base would be most marked in the latter situation.

Basic fractures are produced in two ways—(1) by direct violence; and (2) by indirect violence.

Basic fractures by direct violence, generally regarded as of rare occurrence, include all such fractures as are produced by perforating wounds of the orbit, by bullet wounds through the roof of the mouth, by the driving inwards of the bones of the face, or the driving upwards of the condyles of the jaw, and such also as are produced by falls from a height on to the vertex, the forces then being transmitted through the occipital condyles. I shall, however, endeavour to show that direct violence is responsible for a far greater proportion of all basic fractures.

Basic fractures by indirect violence.—Under this heading are included in most text-books the great majority of basic fractures and various explanatory theories have from time to time been brought forward of which the following are the more important.

1. *Avan's theory of irradiation*, which states that "fractures of the base result as extensions from fractures of the vault, the force following the shortest anatomical route of the base."

2. *The bursting and compression theories.*—The skull is here regarded as a highly elastic sphere, compression of which leads

to diminution in the diameter along the axis of greatest pressure, bulging occurring in other diameters. The bulging exceeding the limits of elasticity a fracture results, the line of fracture varying in different cases. 1. When the lines of fracture run parallel to the direction of the compressing force the bone bursts open along the convexity (bursting fractures). 2. When the lines of fracture run at right angles to the direction of the compressing force a fracture by "compression" is said to result (compression fractures). These theories are partly based on experiments which have been carried out by inclosing a skull in a tight-fitting box and subjecting the box to a compression force sufficient to produce a fracture. It is obvious, however, that in practice the very great majority of basic fractures are produced in quite a different manner, since over 90 per cent. result either from falls from a height, the front, sides, or back of the head coming into violent contact with some resisting object, or else from blows applied directly to the various parts of the skull. In these cases there can be no question of compression. The elastic properties of the skull also are much exaggerated and the peculiar formation of the base, with its greatly varying areas of resistance, is not sufficiently taken into account. The skull, also, can in no sense be regarded as a sphere, since the vault forms less than two-thirds of a complete sphere, the base passing inwards from the lower limits of this segment in a more or less horizontal direction. Bilateral compression never results in a fracture which runs at right angles to the axis of compression, for the fracture passes always directly between the two compressed areas, modified only by the varying resistance offered to it in its transbasic course. In bilateral compression, also, a "bursting" fracture of the vault, the vault being the only "*convexity*," is never seen. If a vault fracture be present it is seen merely as a fissured fracture extending up from the region of the applied force and gradully tailing off towards the summit of the vertex. Further, I shall show that the lines of fracture proceed across the base always in the direction of the applied force, modified again only by the resistance offered by the stronger and weaker basic regions. These facts, and my own experience, render, in my opinion, the above theories quite untenable.

3. "*Contrecoup*" *theory*.—Fractures of the base alone are occasionally seen with direct evidence that the blow was received on the vault. Such cases have given rise to this theory which states that from the point struck a wave is transmitted through the semifluid brain, producing a fracture at some more distant point. Helferich, for instance, maintains that isolated fractures of the orbital roof, and more rarely of other

parts of the base are produced by the influence of hydrostatic pressure. This theory is exposed to all experience in the mechanism of basic fractures and the peculiar fractures brought forward as examples are capable of a simpler explanation. The base being undoubtedly the weakest part of the skull fractures in this situation, when extending from the vault, frequently show greater comminution in the basic region than in the vault. A blow on the vault, therefore, may not suffice to produce a local fracture and yet the forces, when transmitted to any specially weak basic area, may there be strong enough to produce a fracture. Such specially weak basic areas are supplied by the orbital plates of the frontal bones and by the thin cerebellar fossa walls. In proof of this it is merely necessary to state that it is a common occurrence to find that as the result of a blow on the vertical frontal plate the orbital plates are comminuted, the vertical plate remaining uninjured, or at most the seat of a small fissured fracture. I show on the screen two cases, one a fracture of the cribriform plate of the ethmoid without any fracture of the vertical plate of the frontal bone, and the other a fracture of the posterior fossa which passed across the foramen magnum and tailed off on the dorsum ephippii, the resistance offered being too great. The forces, however, when conducted to the thin right orbital plate, still retained sufficient energy to produce a comminuted fracture in that region (Fig. 1, B). It will here be in place to draw attention to the extreme friability of the orbital plates of the frontal bone, whilst the upward convexity of the orbital roof probably accounts for the fact that when that area is comminuted the fragments are practically always displaced upwards, the peri-orbital fat projecting through the gaps into the cranial cavity.

To arrive at any definite conclusions with regard to the mechanism of basic fractures it is necessary always (1) to take a careful history of the accident so as to determine in what manner the injury was received and also what part of the skull was struck; and (2) to compare the result obtained with all visible external signs of injury. A careful examination of a large number of cases of fractures of both vault and of base showed that in from 30 to 40 per cent. of cases the evidence was clear that the basic fracture resulted from, and extended from, a severe vault fracture. These cases are therefore examples of Aran's "irradiation" theory. This theory, however, further states that the fracture follows the shortest anatomical route to the base, whilst such is not the case, as (1) the line corresponds to the direction of the initial force, and (2) the line of fracture is markedly influenced by the resistance offered to it in its basic course, picking out, usually, all the

weaker spots and avoiding the stronger, and it is only in the most severe cases that a fracture surmounts all obstacles and proceeds to the base directly in the line of the applied force. In over 60 per cent. of cases the injury was received (1) in front, over the lower frontal region, supra-orbital ridges, and external angular frontal process; (2) at the sides, over the auricular and mastoid region; and (3) at the back, over the region of the superior curved line of the occipital bone and the occipital protuberance. In all these cases, therefore, the injury was inflicted at or near the level of the base of the skull. The resultant fracture in all these cases was therefore a basic fracture by direct violence, the fracture passing inwards across the base and splitting the base in the same way as a chisel splits a board of wood, the grain of which may be regarded as representing the weaker lines and any intervening knot as representing the resistance offered by any strong bony buttress momentarily turning aside the force which again later passes onwards, parallel to the original direction, but not necessarily in the same straight line (Fig. 1, c). A blow in this region will tend primarily to involve the weaker area, the base, and secondarily to extend upwards on to the vault, many of the fractures in which situation result as extensions from blows delivered around the basic circumference. The statement, therefore, that all basic fractures result as extensions from the vault (Aran) is analogous to putting the cart before the horse, since many of these vault fractures result as extensions from the basic region.

There is still, however, another very important anatomical fact to consider and one which must necessarily exercise great influence on all basic fractures which result from falls or blows on the lateral aspect of the head and in most fractures produced by bilateral compression. The skull may be regarded as consisting of two parts, one part lying anterior to the condyles and the other posterior to, and including, the condyles, this latter part, owing to the vetebral attachment, being firm and fixed (Fig. 1, d). As the result of a blow on the antero-lateral region of the head this anterior segment tends to be split off from the more fixed posterior part. If, again, in a fall from a height the post-mastoid region should strike the ground there is in that region a rebound force which acts in the opposite direction, whilst the anterior segment momentarily travels in the original direction; the two segments are consequently influenced for a brief period by forces acting in opposite directions with the inevitable result that they tend to be split one from the other. It is now necessary to point out that between the two segments there exist an exceedingly weak line, this line passing

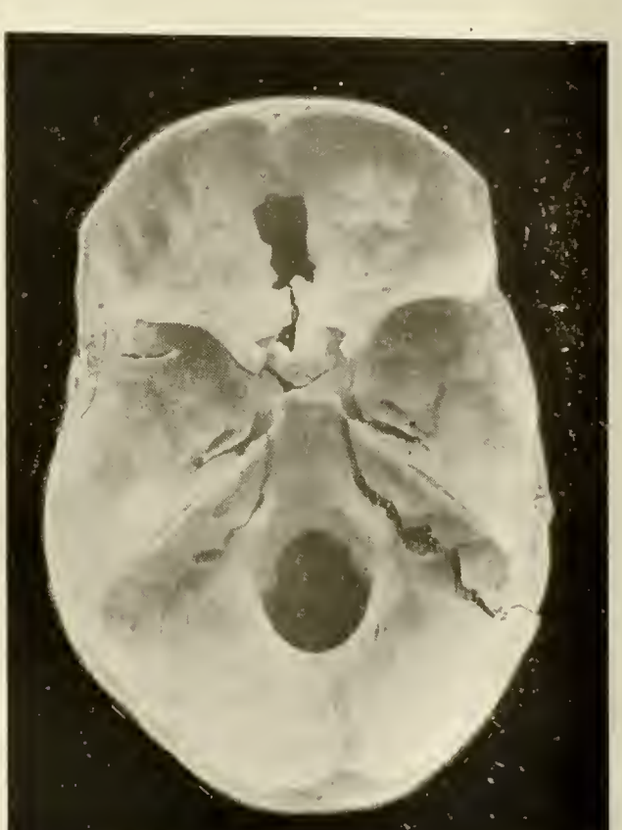
from one external auditory meatus to the other, involving in its course both middle ears, both petro-sphenoidal sutures, with the sphenoidal sinus as a connecting link in the middle line (Fig. 1, D). Such a fracture, as is indicated in the illustration, exemplifies a common and typical basic fracture and one which is most frequently seen when the accident is the result of a fall from a height. It is usual to find that the fracture extends up, on one or on both sides, on to the vault and occasionally this vault extension is so marked that the whole skull is practically divided into two parts, merely united by soft structures. More than once I have been able to elicit crepitus on the cadaver by rubbing the anterior and posterior segments together.

Collecting now the facts, so far as I have gone, I am of opinion: (1) that the bursting and compressing theory and the contrecoup theory are both untenable; (2) that Aran's irradiation theory, with certain modifications, accounts satisfactorily for those basic fractures which result from blows on the vertex; and (3) that most basic fractures result from forces applied directly around the basic level and that these fractures are the result of a splitting force, the line of fracture tending to travel across the base, parallel to the original direction, but not necessarily in the same straight line. It now remains to consider other factors which may exercise great influence on the actual direction of the fracture.

1. *The influence of sutures.*—Complete maceration of the skull is always necessary in estimating how far the various sutures of vault and base are affected in severe head injuries, for the dura mater, dipping down in these situations, usually completely obscures the lesion unless the degree of separation is marked. This injury is generally regarded as of rare occurrence and it is said by Morgagni to be most prevalent in the young. An examination of a large number of macerated skulls shows, however, (1) that separation along certain sutural lines is very common; (2) that this separation is most common in middle life; that not only does a suture tend to deaden a force, but if a suture lies in the path of a fracture, the forces tend preferably to follow that suture, even though the original direction may be slightly altered in so doing; (4) that in the more simple sutures the separation follows exactly the irregular sutural line, whilst in the more intricate sutures the line of separation passes along the suture, regardless of the finer dentations; and (5) that sutural separation, without any further basic lesion, is possible but of rare occurrence. The masto-occipital, petro-occipital, and lambdoid sutures are most commonly implicated (Fig. 2).

2. *The influence of the air sinuses.*—(*a*) The sphenoidal sinus, formed by the hollowing out of the body of the sphenoid, varies greatly in size. In the great majority of cases it extends backwards nearly as far as the occipito-sphenoidal junction, whilst laterally it is separated by a thin plate of bone from the cavernous sinus. The roof and floor are usually paper-like in density, whilst the latter also assists in the formation of the naso-pharyngeal roof. The sinus, therefore, is bounded on all sides by a mere shell of bone

FIG. 2.



Showing an extensive fracture of the sphenoidal body, with marked separation along the line of the masto-occipital and petro-occipital sutures.

and consequently there exists in the very centre of the base an exceedingly weak area, an area, therefore, which would naturally be involved in a very large proportion of all basic fractures. From my own experience this sinus was involved in 70 per cent. of cases, the comminution of roof and floor being in some cases so excessive that a large probe could be passed from the naso-pharynx into the cranial cavity so affording free means of ingress to micro-organisms (Fig. 2). Severe hæmorrhage may result from concomitant injury to the cavernous sinus and to the internal carotid artery, with

furious bleeding into the naso-pharynx and nose. Fig. 1, E, shows the great liability of the sinus region to injury, as (1) all fractures from one middle fossa to the other pass across the sinus (*a*, *a*); (2) all fractures from one middle to the opposite anterior fossa also involve this region (*a*, *b*); and (3) most fractures of the anterior fossa or of the middle fossa tend to terminate in the sphenoidal body (*c*, *c'*, *c''*).

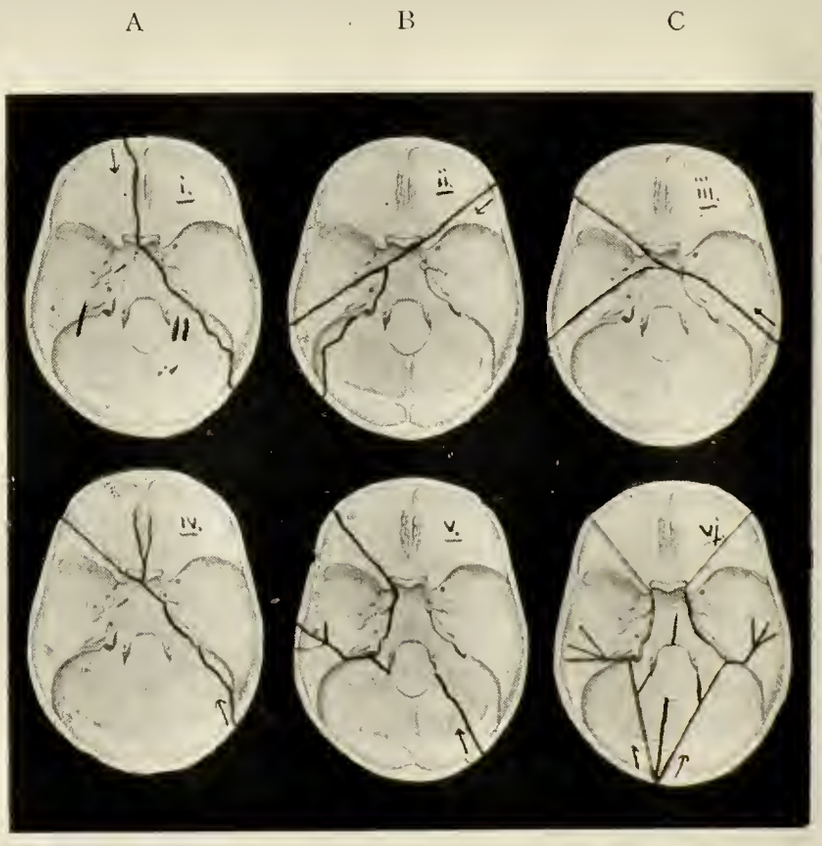
(*b*) Up to the age of puberty the frontal sinus is either absent or represented merely by a small air-cell, but after puberty it develops rapidly in size and extent. This fact is of importance in estimating the gravity of a fracture in the supra-orbital region at the different ages. The two sinuses are also generally asymmetrical and not infrequently the sinus extends far backwards into the orbital roof. The inner boundaries are often exceedingly thin and friable, whilst the outer wall consists of fairly dense bone, and consequently a fracture of the outer wall is almost invariably accompanied by a more severe and frequently comminuted fracture of the inner wall, or, in other words, by a fracture of the anterior fossa (Fig. 14). The more prominent immediate resulting symptoms are: (1) hæmorrhage into the orbit and eyelids; (2) hæmorrhage into the nose; and (3) traumatic emphysema, from the escape of air into the surrounding tissues, secondary to injudicious blowing of the nose, &c. The air in the cellular tissues may spread widely over the vault and face and into the retro- and peri-orbital tissues, with great swelling of the eyelids and proptosis of the eye. When localised the condition is known as "traumatic pneumatocele."

With regard to later complications it must be borne in mind that the more extensive fracture of the inner wall affords a mode of ingress for septic organisms with resultant extradural abscess or meningitis. It is therefore advisable when suppuration in the external wound occurs to chisel away or to trephine a portion of the outer limiting wall in the most dependent part in order to establish free and thorough drainage, the normal channel of exit into the middle meatus of the nose being usually blocked by swollen and lacerated mucous membrane or by blood clot. Unless some such treatment is adopted empyema of the sinus will probably result and death ensue from direct meningeal infection.

The auditory region.—I shall discuss later the great liability of this region to injury and will merely state here that this weakened area is involved in the great majority of middle fossa fractures. The exact lines of the fracture, the resultant hæmorrhage, the involvement of the seventh and eighth nerves, and other features will all be alluded to under their separate headings.

The basic foramina.—Battle, the only authority on skull fractures who enters into detail with regard to the influence of the foramina on the direction of a fracture, states that "the line of fracture is usually arrested if it meets with one of the foramina in its course, especially if the foramen be a large one." A careful examination of macerated skulls shows, however, that this view is erroneous; Fig. 1, F illustrates three separate cases, chosen from numerous others, in which the fracture after reaching the margin of the foramen magnum was

FIG. 3.



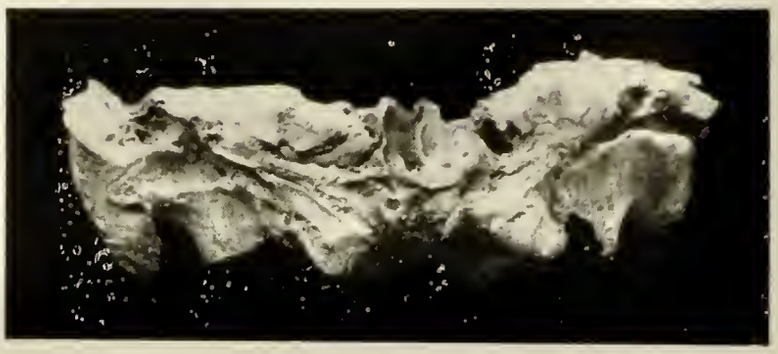
Showing lines of forces as applied and the direction of fractures resulting therefrom.

again continued on the other side of the foramen. The fracture, after traversing the thin cerebellar fossa, usually strikes the foramen magnum just posterior to the condyle and starts again at a corresponding point behind the opposite condyle, tending then to pass onwards parallel to the original direction but not in the same straight line. More rarely a fracture on reaching the foramen magnum is continued on the opposite side in the same straight line. This condition is, however, exceptional and I have only met with two such cases,

both of which are shown in the illustration (Fig. 1, F). Turning from this foramen to others it is exceedingly common to find fractures traversing the foramen lacerum anterium, the foramen lacerum medium, or any other foramen that may lie in the chosen paths of basic fractures. Having now drawn attention to some of the more important anatomical features which exercise greater or lesser influence on the direction of a basic fracture, I will point out that by a careful comparison of the direction of the applied force and the point of application it is possible in the greater number of cases to arrive at conclusions guiding one in estimating the probable line of fracture in any given case (Fig. 3).

Direction, &c., of the Applied Force.	*Resultant Fracture*
i. Force applied to median frontal region. (Fig. 3, A.)	The fracture passes backwards from the perpendicular plate of the frontal bone to the cribriform plate of the ethmoid, thence between the optic foramina to the body of the sphenoid, the thin sinus roof being usually comminuted. From there the fracture diverges to the opposite side, and tearing off the posterior clinoid process passes along the petro-occipital suture to the jugular foramen, being again continued on the other side of that foramen along the masto-occipital suture, and so again to the vault.
ii. Force applied to the lateral frontal region, in the situation of the external angular frontal process. (Fig. 3, B.)	The fracture passes across the anterior fossa towards the sphenoidal fissure, tearing away the anterior clinoid process, and again comminutes the roof of the sphenoidal sinus. Progressing onwards the posterior clinoid process is torn away and the fracture then passes either along the anterior part of the petrous bone at its junction with the greater wing of the sphenoid towards the opposite middle and external ears, or along the petro-occipital suture to the jugular foramen and continued along the masto-occipital suture, as in the previous case.
iii. Force applied to the region of the external ear. (Fig. 3, C.)	The fracture passes across the roof of the bony auditory meatus towards the junction of the anterior and inner walls of the middle ear, the membrane and ossicles undergoing a varying amount of destruction and displacement. The fracture is then continued across the tegmen, which is in most cases more or less comminuted, and after following the region of the petro-sphenoidal suture it reaches the foramen lacerum medium, being continued again on the opposite side of that foramen to the sphenoidal body. Thence it pursues one of two courses. Most commonly the fracture passes obliquely backwards to the opposite middle and external ears, following a course similar to that already indicated. (Fig. 4.)

Fig. 4.



Showing portion of the base of the skull with a fracture involving both auditory regions, and the sphenoidal sinus. On the left side of the illustration, the geniculate ganglion of the facial nerve is seen; other structures are exposed as described in the text.

In such cases the basic fracture may extend on each side up on to the vault in such a manner that the two segments are merely united by soft parts and an examination of the anterior aspect of the posterior fragment reveals further that the fracture passes just anterior to the geniculate ganglion of the facial nerve, which ganglion usually lies exposed. Its petrosal branches are, however, generally torn. The fracture also passes anterior to the Eustachian tube and anterior to the horizontal part of the internal carotid artery. In passing from without inwards, therefore, the following structures are to be noticed on the anterior aspect of the posterior fragment; the posterior half of the external auditory meatus, the mastoid antrum, the lacerated membrane and ossicles, the posterior two-thirds of the middle ear, the geniculate ganglion of the facial nerve, the Eustachian tube, the horizontal part of the internal carotid artery, the Gasserian ganglion, and the posterior part of the sphenoidal sinus in the middle line (Fig. 4). After reaching the sphenoidal body, the alternative course for the fracture to pursue is to pass towards the opposite sphenoidal fissure, and tearing off the corresponding anterior clinoid process, to pass onwards across the anterior fossa, parallel to the original direction but not in the same straight line.

iv. Force applied to the mastoid region.

(Fig. 3, D.)

The fracture follows the occipito-mastoid suture to the jugular foramen, and is again continued on the opposite side of this foramen, following the petro-occipital suture towards the apex of the petrous bone. Tearing off the posterior clinoid process, the fracture now passes across the sphenoidal body to the sphenoidal fissure of the opposite side, and

so across the anterior fossa, parallel to the original direction. It is especially common in this particular variety of fracture to find fissures diverging from the region of the sphenoidal body forwards towards the cribriform plate the fissures passing mainly between the optic foramina and not usually directly involving those foramina. This fracture is also peculiar in so much that when the degree of separation along the occipito-mastoid suture is excessive there is a special liability to a tearing of the dura mater with injury to the sinus wall just as the sinus begins to dip downwards and inwards (Figs. 2 and 13).

FIG. 5.

Showing fracture passing through the left cerebellar fossa, and continued on the opposite side of the foramen magnum across the right petrous bone.

v. Force applied to the lateral occipital region.
(Fig. 3, E.)

The fracture passes across the thin cerebellar wall. usually leading to some degree of comminution, and strikes the foramen magnum just posterior to the condyle. starting again at a similar position on the other side of the foramen and passing to the outer margin of the jugular foramen (Figs. 5 and 15) Two courses, again, are now available, for the fracture may either cut across the body of the petrous bone, external to the internal auditory meatus, finally comminuting the roof of the

middle ear, or else the fracture may be continued from the inner part of the jugular foramen along the petro-occipital suture towards the apex of the petrous bone and the foramen lacerum medium. It usually then involves the corresponding sphenoidal fissure and passes across the anterior fossa, parallel to the original direction.

vi. Force applied to the posterior occipital region.

(Fig. 3, F.)

The resultant fracture varies according to the direction of the applied force. A force when applied to the posterior occipital region at right angles to the transverse axis of the skull results in a fracture which, on reaching the posterior margin of the foramen magnum, is continued on again on the opposite side of the foramen along the dorsum ephippii. When the force is more oblique in direction the fracture traverses the thin cerebellar fossæ to the outer margin of the jugular foramen and then follows one of the two courses indicated in the previous case. More commonly the fracture cuts across the petrous bone. An offshoot is frequently seen which passes to the foramen magnum just posterior to the condyles. The cerebellar wall is frequently comminuted and the lateral sinus may be torn in the line of the fracture.

Basic fractures, therefore, follow certain definite paths, the transbasic course varying according to the direction of the applied force and the point of application. These plans were worked out in the post-mortem room and were subsequently put to a practical use with the result that a correct diagnosis as to the path of the fracture was made in almost every case. It must be, however, clearly understood that the extent of the fracture, whether it completely traverses the base from side to side or not, depends on the amount of force used and the resistance offered, for bases, as well as vaults, vary greatly in density. To every rule also there must be exceptions and occasionally one may find some peculiar basic fracture which seems to follow no rule, whilst in other cases the force may be so excessive that the fracture crosses the base regardless of all obstacles. I venture to assert, however, that the lines of fracture indicated will in the great majority of cases be found to be correct. Finally, with regard to fractures produced by bilateral compression the line of fracture always tends to run between the two compressed areas, modified only by the relative strength of the intervening areas.

Fractures of the vault.—Fractures of the vault may be limited to that region or they may coexist with a basic fracture. Evidence has been brought forward under "fractures of the base" to show that in the latter class of cases the vault fracture

frequently results as an extension upwards from the basic circumference. Whether, as the result of a blow on the vault, the fracture remains limited to that region or extends downwards also to the base depends mainly on the degree of force used and the direction in which that force is applied. The greater the force the greater is the tendency to vault limitation, whilst, on the other hand, a force applied obliquely downwards on to the skull surface usually results in an extension towards the basic area, not, however, following the shortest anatomical route to the base, as Aran indicated, but guided to the base by the direction of the applied force and by the resistance offered to it in its course. The fracture passes preferably between the various bony buttresses which pass up from base to vault, except in those cases where, owing to greater violence, it passes to its termination regardless of the obstructions which may lie in the path. Lastly, fractures of the vault, produced by falls from a height on to the summit or on to the side of the head, are usually complicated by a concomitant basic fracture, the weight of the falling body being transmitted through the condyles, greatly influencing the extent and direction of the forces acting. The varieties of vault fracture are of such text-book prominence that I propose to confine myself to three points only.

1. *Fractures of the external table.*—The possibility of a fracture of the external table without any injury to the internal table was up to recent years a matter for dispute. The late war in South Africa has, however, conclusively proved the existence of such a fracture. This "gutter-shaped" fracture usually results from a glancing or spent bullet. Such fractures are, nevertheless, of such rare occurrence and of such difficult diagnosis that they form no exception to the rule that exploration and trephining should be carried out when there is any suspicion that a more extensive injury to the internal table may also be present.

2. *Fractures of the internal table.*—"When a foreign body passes completely through any part of the skull—it matters not what the direction may be—the aperture of exit is always larger than the aperture of entry." Such was the law enunciated by Teevan in 1864 and explained by him as follows: "The aperture of entry is caused by the penetrating body only, whilst the aperture of exit is larger, inasmuch as it is made by the penetrating body plus the fragments of bone driven out of the proximal table and diploe." Previously to this theory the explanation offered was that the internal table was the more brittle. Teevan proved that this was not the case as by firing a bullet from inside the skull the wound of exit was again greater than the wound of entry. It was also

proved that whether the inside or outside of a skull be struck fracture of the distal table only, without any injury to the proximal table, can be produced in either case. This is, as Teevan pointed out, in obedience to the law that "when pressure is applied to a body, when it breaks the fracture commences on the line of extension, not that of compression." Fractures of the internal table are most common and most extensive in those regions where the diploic tissue is most abundant—e.g., the mid-parietal and anterior frontal regions. Too much stress, therefore, cannot be laid on the fact that (1) fractures of the internal table only may exist without any visible or palpable injury to the external table; and (2) that when the external table is fractured there is in nearly every case a more extensive fracture of the internal table. The depressed internal table may give rise to many complications, of which the following are the more important.

(1) *Immediate.*	(2) *Remote.*
A. Injury to the membranes, venous sinuses, and meningeal arteries.	*A*. Meningeal thickening, chronic cerebritis, and arachnoid cysts with consequent localised cephalalgia, Jacksonian epilepsy, and insanity.
B. Injury to the brain substance.	*B*. Meningitis, cerebral abscess, &c. (when compound).

Some of the complications and the question of treatment will be alluded to later.

3. *Explosive fractures.*—The passage of a bullet through the brain leads occasionally to such intracranial disturbance of the fluid and more solid contents that the resultant explosive force, combined with the forces acting on the skull from the impact of the bullet, produces a peculiar fracture which I may call an "explosive fracture." More commonly the sutures give way, especially along the line of the sagittal suture, but in more rare cases the vault may be more or less completely lifted off from the base. This unique specimen which I show you is an example of this latter variety. The bullet, fired from a pistol held close to the right temporal region, passed through the brain and impinged against the opposite parietal region, producing an "elevated" fracture, the external table being more extensively damaged than the internal. In spite of an abnormally massive skull an irregular fracture extended completely round the skull, except for about two and a half inches in the right occipital region, lifting the vault from the base like a trap-door. There was no basic fracture. (Fig. 6.)

Fractures in children.—In comparing a child's skull with that of the adult it can at once be seen that there are certain peculiarities in the former which must influence the direction

and the extent of a fracture. 1. The bones of the vault are elastic and pliable and consequently a blow may lead to a bending-in, temporary or permanent, without any actual fracture. Dense fibrous tissue also intervenes between the bones, tending to limit the fracture to the bone struck, extensions to the basic regions being comparatively rare. 2. The diploic tissue is practically absent and consequently fractures of the internal or external tables only are almost

FIG. 6.

Explosive fracture caused by a pistol-shot in the right temporal region.

unknown. 3. The air sinuses are non-existent or small. It is especially important to bear this in mind in estimating the gravity of severe fractures in the anterior frontal region. The greater adherence of the dura mater in the child is said to prevent the formation of any extensive extradural clot. There can be no question that middle meningeal hæmorrhage in children is rare, but I put this down, not to the supposed greater adherence of the dura mater, but to the fact that, owing to the elasticity of the skull

and the absence of bony spicules from the internal table, the artery usually escapes laceration, being more commonly pushed aside or compressed. Bruising and superficial laceration of the brain are, however, relatively more common in children than in adults, owing to the greater delicacy in structure of the arachnoid, pia mater, and cortex. A comparatively slight blow leads consequently to subpial and subarachnoid hæmorrhages, resulting in violent convulsions when situated over the Rolandic area. Fractures of the base, though obviously of less frequent occurrence, in their direction and extent follow the rules already put forward under "fractures in adults." The presence of the petro squamous suture makes more grave the prognosis of a fracture, involving the middle ear and being followed by suppuration.

Relative frequency of fractures of the anterior, middle, and posterior fossæ.—A basic fracture may be limited to one fossa only or all three may be involved at one and the same time, whilst in rare cases the anterior and posterior fossæ may be fractured, the intervening middle fossa remaining uninjured. Fractures implicating the anterior and middle fossæ are almost invariably accompanied by definite symptoms, rendering the diagnosis fairly certain, whilst fractures of the posterior fossa are frequently overlooked owing to the absence of definite clinical symptoms, and it is of common occurrence to find at the post-mortem examination a fracture in this situation the existence of which had not previously been suspected. It is probable that fractures of the middle fossa are the most common and fractures of the posterior fossa the most rare.

Mortality according to the fossa implicated.—In endeavouring to estimate the mortality according to the fossa implicated the same difficulties arise owing to the uncertainty of the diagnosis of posterior fossa fractures. There are, first of all, dangers peculiar to each fossa. 1. Anterior. Septic infection from the nose, ethmoid cells, &c. 2. Middle. Septic infection from the naso-pharynx and ear, injury to the middle meningeal and internal carotid arteries. 3. Posterior. Injury to the venous sinuses. Fractures of the middle fossa, therefore, are the most dangerous from local complications. There is, however, a grave complication which is common to all fossæ, injury to the brain itself, and since the vital centres are grouped posteriorly in the region of the fourth ventricle the prognosis of fractures of the posterior fossa is correspondingly rendered more grave. Probably the mortality increases from before backwards, fractures of the anterior fossa being certainly the least dangerous, with but little to choose between a fracture involving the middle fossa and one

implicating the posterior fossa. The general mortality as given by different authorities does not vary very much. Battle from 168 cases puts the general mortality at 32 per cent. This is, according to my experience, too low, for in 215 cases 89 died and 116 recovered. The mortality equalled 44 per cent. Fractures of the base occur at all ages of life, the death-rate being highest at the two extremes of life, from 50 to 60 per cent. under the age of five years, and from 60 to 70 per cent. after the age of 70 years.

Mortality of vault fractures, with and without depression.—The general mortality of vault fractures, with or without depression, is from 20 to 25 per cent., the death-rate now being lowest in children and highest in old age.

There are still some surgeons who refuse to believe that a patient can recover from a severe basic fracture. Clinical experience, however, shows again and again that even after the most extensive injuries patients may completely recover, and this is especially the case in young adults. As an example I will briefly narrate one case. A boy, aged 15 years, was hanging by his feet from a wharf when a barge, swinging inwards, crushed his head against one of the piles. When admitted he was collapsed and pulseless and bleeding profusely from both ears, from the nose, and from the mouth. Both tympanic membranes were lacerated at the upper and anterior part and subsequently marked facial paralysis was seen on one side. One pupil was widely dilated and the other was contracted to a pin-point, and added to this there was marked internal strabismus of one eye with proptosis. In spite of all these troubles the boy made a rapid recovery, all symptoms disappearing except the squint. A more typical picture one could not desire and the symptoms all indicated one of the most severe basic fractures —that fracture which I call "the typical basic fracture"—as it undoubtedly passed from one auditory meatus to the other (Fig. 4), involving both middle ears and the sphenoidal sinus in the middle line. The line of fracture passed just in front of the genu of one facial nerve and from the sphenoidal region there was an extension forwards to the sphenoidal fissure, thus accounting for the orbital symptoms. The sixth nerve was probably injured, as it laterally grooves the dorsum ephippii and, as I shall point out under "nerve implication," injuries of the sixth nerve in that situation frequently lead to a permanent paralysis.

Though there are but few who refuse to believe in the possibility of recovery in such cases, yet there are many who regard a posterior fossa fracture as necessarily fatal. Such a view is hard to refute by a mere recitation of cases, since these

fractures are conspicuous by their lack of clinical symptoms. The illustration, however, is that of a united fracture of the posterior fossa and it exemplifies well certain peculiarities common to all united basic fractures (Fig. 7). The new bone formation is poor in quality and in quantity, being rough and porous, and almost limited to the inner aspect of the skull. On the outside the fissure remains quite definite, the edges being slightly rounded off with scattered bridges of new bone formation. The dura mater is generally firmly adherent to the rough new bone and also thickened and discoloured.

Fig. 7.

A united fracture of the posterior fossa of the skull.

The medico-legal aspect of skull fractures.—The next illustration is of great interest on account of its medico-legal importance. The man from whom the specimen was obtained was seen by several witnesses to commit suicide in a state of suicidal frenzy by striking himself repeatedly on the vertex of the skull with a dumb-bell. The evidence of repeated blows is borne out by the appearance of the vault. A central hole, nearly circular in shape, is surrounded by numerous semi-circular dents and fissures, showing that several blows were inflicted (Fig. 8). Those fragments which were not driven into the brain were torn out by the patient before he became unconscious. The greater destruction of the internal table is also of interest. The position of the fracture, the evidence of repeated blows, and the nature of the implement used would in the absence of reliable witnesses have been sufficient to lead to a verdict of " wilful murder."

Two other cases of equal medico-legal interest are reported

by Mr. R. Withers, of Plymouth. The first case was that of a man who was admitted into the Tuapeka Goldfields Hospital, New Zealand, 25 years ago, suffering from a comminuted fracture of the frontal bone, exposing the dura mater. He was found unconscious in a hut with a blood-stained tomahawk lying by his side. His mate was arrested, but fortunately for him the injured man recovered consciousness after ten days and made the following statement. Having collected sufficient gold he had been deputed by his mate to get stores from the nearest town. There he had spent the money in a prolonged debauch and on his way home an angel

Fig. 8.

A fracture of the vertex of the skull from repeated blows.

appeared to him and pointed out to him the iniquity of his conduct in thus defrauding his mate. In consequence of this and in order to expiate his offence, on his arrival at the hut he knelt down on the floor and proceeded to smash in his forehead with the butt end of the tomahawk, continuing his efforts till he lost consciousness.

The second case was that of a Kanaka on a sugar estate in Australia. This man was found lying in the hut of another Kanaka with his forehead crushed in and a hatchet beside his body. He remained insensible for several days but eventually recovered and stated then that his injuries were self-inflicted and that he went to the other man's hut so that suspicion of murder should rest upon him.

LECTURE II.

Delivered on March 2nd.

Hæmorrhages.

Mr. President and Gentlemen,

The various hæmorrhages that may result in skull fractures may be first classified into two main groups: (1) external hæmorrhages and (2) internal hæmorrhages. The first of the two groups may be subdivided into those hæmorrhages which result from basic fractures and those which are secondary to a fracture of the vault. The various extravasations into the tissues of the scalp would be included in the second subdivision, but as such hæmorrhages present but few points of new interest I do not propose, therefore, to discuss them further.

1. *External Hæmorrhages in Basic Fractures.*

Hæmorrhage from the ear.—The great majority of fractures which involve the middle fossa of the skull originate laterally at the external auditory meatus and pass inwards from that region towards the sphenoidal sinus. Both the roof and floor of the external auditory meatus may be involved, but the fracture is generally most marked along the roof. From this region the fracture passes towards the anterior and internal angle of the middle ear, the membrane undergoing a variable degree of destruction in the path of the fracture. The degree of membrane laceration corresponds to the severity of the fracture and the exact nature of the forces producing the fracture. For instance, if the fracture extends from the vault downwards to the base (Figs. 9 and 10) the membrane is generally torn in its upper and anterior part only, whilst if the fracture results from a direct blow on the auricular region the osseous injury is more extensive and the degree of laceration is added to by the forcible in-driving of air, whilst in the most severe cases the drum destruction is complete. The malleus and the incus undergo varying dislocations, but the stapes rarely suffers any change in position. The blood which escapes from the ear will be seen to be derived from the lacerated cuticle lining the roof of the

external meatus and also from a deeper region. Hæmorrhage is sometimes so profuse that one is quite unable to determine the exact source, but in favourable cases an otoscopic examination will reveal that the blood comes from the anterior and upper region of the tympanum, not so much from the torn drum itself but welling up fast through the gap in the drum. Post-mortem examination in such cases shows that the middle ear is full of blood derived from those torn tympanic vessels which, supplying the mucous lining and bony walls of the middle ear, lie in the path of the fracture.

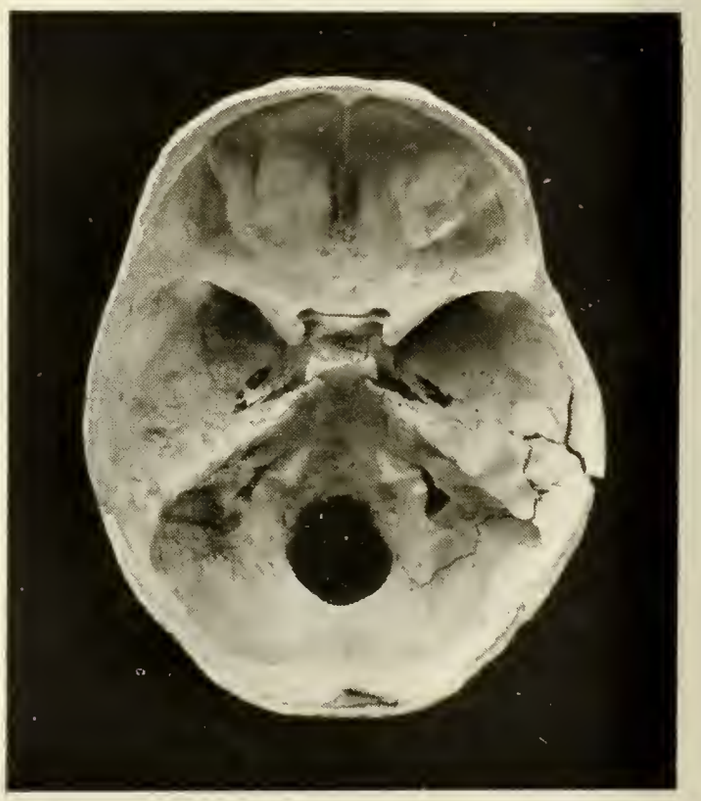
Fig. 9.

A fracture extending downwards from the vault to the base and involving the external auditory meatus. The vault has been sawn through horizontally.

The amount of blood lost varies greatly. The escape may be profuse and may be continued for hours and even days, whilst in other cases there may be merely sufficient extravasation to clot and to dry up in the external meatus. I have had the opportunity of seeing several cases that throw light on the main causes which influence the amount of blood lost. The first case which drew my attention to the subject was as follows. A man was admitted with a wound on the right side of the head, the result of a fall, and with profuse hæmorrhage from the right ear, which lasted till death ensued, 14 hours later. The patient presented no symptoms pointing to extradural hæmorrhage, yet at the post-mortem examination a very extensive hæmorrhage from the middle

meningeal artery was found, while the tegmen tympani was markedly comminuted, the blood having a free means of escape into the middle ear and so externally through the lacerated membrana tympani (Fig. 10). On going into the question, in subsequent cases, I observed (1) that hæmorrhage from the middle meningeal artery is very much more common than is generally supposed to be the case, the slighter forms yielding no clinical symptoms; and (2) that the fissures running across

Fig. 10.



Internal view of skull shown in Fig 9. An extensive fracture of the tegmen tympani accompanied by laceration of the posterior branch of the middle meningeal artery.

the tegmen tympani are usually of such a nature that blood can be forced through them into the middle ear.

It should also be pointed out that the extradural extravasation exercises a great mechanical pressure, and that as the extravasation increases so in direct ratio does the pressure exercised by it on the surrounding structures also increase. Relief, therefore, takes place in such cases by the passage of blood into the middle ear and through the torn membrana tympani. I regard, therefore, profuse and continuous hæmorrhage from the ear as very suggestive of an accompanying extradural hæmorrhage and although it is, perhaps, too much to advocate trephination over the artery in all such cases yet

I am strongly of the opinion that such a method is advisable when the general conditions are grave, even in the complete absence of the typical symptoms of middle meningeal hæmorrhage. It is hardly necessary to add that the closure of the external meatus with a plug of gauze kept in position with wool and bandage is the worst possible treatment, and I have seen, at any rate, two cases where such methods quickly resulted in an increase of compression symptoms and in accelerated death. Both these cases presented on later examination all the features I have alluded to. The application of a plug in the meatus may, perhaps, be used for diagnostic purposes, the patient being carefully watched and any alteration in the symptoms pointing to pressure over the motor area promptly treated by trephination.

It will be of interest to narrate briefly a somewhat similar case reported by Chassaignac.[1] A man after falling down an area had continuous hæmorrhage from the ear lasting for three days. He kept to his bed but on the third day, feeling otherwise well, he got up and walked briskly to consult a "wise woman." On returning he complained of fatigue and headache, the hæmorrhage now having ceased. He became delirious and died. The examination showed an extensive fracture of the petrous bone with laceration of the lateral sinus and a great extravasation from a ruptured middle meningeal artery. The blood poured out had escaped freely for three days, the passage had then become blocked, and death resulted. In stating, therefore, that free hæmorrhage from the ear suggests strongly an extradural hæmorrhage from the middle meningeal artery it should be added that the blood may be derived from a lacerated venous sinus, this latter condition being, however, much more rare than the former (Fig. 13).

Hæmorrhage from the ear does not, of course, necessarily imply the existence of a basic fracture. The membrane alone may be ruptured as the result of a blow on the ear. The resultant hæmorrhage is, however, seldom profuse and the absence of any further symptoms will soon clear up the diagnosis. From my own experience, however, I think that too much stress is laid on the fact that hæmorrhage from the ear results from other causes than a basic fracture and I regard this symptom, when secondary to a blow on the head, as practically diagnostic of the particular fracture of the middle fossa to which I have alluded. In some few cases hæmorrhage occurs from both ears, usually as the result of a bilateral compression, the fracture running from one external auditory meatus to the other, involving both

[1] Plaies de la Tête, 1842

tympanic cavities and the body of the sphenoid in the middle line (Fig. 4). In these cases also the fracture frequently extends on either side up on to the vault in such a manner that the skull is practically divided into two parts, merely united by the soft structures. The prognosis in these cases is necessarily grave but the marvellous vitality of young adults enables them to recover even in such adverse circumstances.

Hæmorrhage into the nasal cavity and into the naso-pharynx.—Hæmorrhage into one or both of these cavities is one of the most constant symptoms in fractures of the base, for it occurs in nearly all fractures involving either anterior or middle fossæ. The post-mortem lesions found in these cases are comminuted fractures of the cribriform plate of the ethmoid and of the sphenoidal body (Figs. 14 and 2).

I have already pointed out that most anterior fossa fractures traverse the cribriform plate of the ethmoid, a fairly free hæmorrhage resulting from lacerated ethmoidal arteries, whilst the very great majority of fractures of the middle fossa tend to pass through the sphenoidal body. Now this sphenoidal body, which is in reality one of the weakest of the basic areas, consists of a mere shell of body inclosing the air sinus and bounded laterally by the cavernous sinus, on the outer side of which the internal carotid artery runs. The liability of this region to injury is so marked that although a fracture passing along the anterior border of the petrous bone may tail off before reaching this region, yet in many cases the conducted forces suffice to bring about an extensive fracture on reaching the sphenoidal body. These fractures are generally comminuted, both roof and floor of the sinus being equally damaged in such a manner that a probe may be passed from the cranial cavity into the naso-pharynx below. The comminuted sinus wall punctures or tears the cavernous sinus, profuse hæmorrhage resulting, whilst in the most severe cases the internal carotid artery itself may be completely torn across. In the only two cases that I have seen where the carotid artery was torn blood poured into the naso-pharynx, gushed from the mouth in streams, and death resulted almost immediately (Figs. 11 and 12).

Hæmorrhage into the orbital region.—The results produced are: (1) paralysis of the muscles, both from actual mechanical impediment to movement and from pressure on the nerves supplying the muscles; (2) subconjunctival hæmorrhage; (3) palpebral and peri-palpebral hæmorrhage; and (4) proptosis. The muscular paralyses will be alluded to under "nerve implication"; the other symptoms need, however, further reference.

Subconjunctival hæmorrhage.—Hæmorrhage beneath the conjunctiva nearly always makes its appearance at the outer canthus of the eye, progressing inwards towards the corneo-scleral margin. In the more marked cases the extravasation may completely surround the cornea, bulging forwards so as almost completely to obliterate the field of vision. Even in these cases, however, the hæmorrhage is most extensive at the outer canthus, When combined with some degree of proptosis, the probable fracture present is one which involves the region of the sphenoidal fissure, with implication of the anterior part of the cavernous sinus. When no proptosis is present the fracture is commonly due to a blow in the temporal region involving the outer orbital wall.

Palpebral and peri-palpebral hæmorrhage.—Hæmorrhage into the eyelids and into the loose connective tissue surrounding the lids is seen in most cases of anterior fossa fractures. It frequently coexists with subconjunctival hæmorrhage but the two are not necessarily present together. It differs from subconjunctival hæmorrhage in its site of early origin, for palpebral hæmorrhage nearly always begins at the inner angle of the eye, progressing outwards into the tissues of the upper and lower lids and into the surrounding regions. The extravasated blood may be wholly anterior to the tarsal and ligamentous structures of the lids, in which case the fracture usually involves the perpendicular plate of the frontal bone only. More commonly, the fracture extends along the orbital roof, perhaps involving the sphenoidal sinus, in which case palpebral hæmorrhage, subconjuntival hæmorrhage, and proptosis may all three be present together.

Proptosis.—The time at which proptosis first becomes evident and the degree to which it progresses are both very variable. One can, however, tabulate these cases roughly into three main groups and from them draw certain conclusions as to the probable nature of the injury present.

1. Proptosis severe, appearing almost at once, and progressive.	1. A severe fracture in the region of the sphenoidal fissure and cavernous sinus with extravasation of blood forwards into the retro-ocular tissues. Thrombosis of the cavernous sinus may coexist and in the most severe and progressive cases there may be some damage to the internal carotid artery.
2. Proptosis moderate and appearing after a few hours.	2. A fracture involving the orbital walls, the extravasated blood being derived from lacerated ophthalmic vessels.
3. Proptosis appearing days or weeks after the injury and usually steadily progressive.	3, A fracture involving the region of the cavernous sinus with injury to the internal carotid artery and with the formation of a fistulous communication between the sinus and the artery.

I have alluded several times to the sphenoidal fissure as a region which, when involved, may result in fairly extensive hæmorrhage. I should remind you, perhaps, that the orbital branch of the middle meningeal artery runs forwards to the orbit at the outer angle of the fissure and that this vessel is commonly injured in all fractures involving this region, adding, therefore, to the blood extravasation which results from fractures involving this region.

Hæmorrhage into the mastoid and occipital regions.—In fractures of the posterior fossa the blood poured out has but little chance of making itself evident owing to the dense mass of muscles and fasciæ which overlie that fossa. The resistance offered by these structures tends to confine the extravasated blood to the cerebellar fossæ, adding consequently to the already grave prognosis of fractures in this region. Sometimes careful palpation will detect a "doughy" or "boggy" condition of the tissues and ecchymosis may appear later from the extravasation of blood upwards on to the vault and downwards into the neck. A peculiar ecchymotic patch is frequently seen, appearing first in front of the apex of the mastoid process and travelling upwards in a curved direction, concavity forwards, following the outline of the external ear. This ecchymosis is said to result from the tracking of blood upwards along the course of the posterior auricular artery. In all the cases that I have seen this condition was associated with a separation along the occipito-mastoid suture and although this suture and the line of the hæmorrhage do not quite coincide there must, I think, be some connexion between the two (Fig. 2). Before passing on to the subject of intracranial hæmorrhages I wish to return to the subject of

"*Orbital aneurysm.*"—The first complete account of this complication was given by Rivington in 1875.[2] I have collected 20 further cases, in all of which the history and other symptoms strongly indicated the previous existence of a basic fracture. 16 of these cases were males and four were females. The average age of the males was 22 years and that of the females was $33\frac{1}{2}$ years. The injury consisted of blows or falls on the head in 18 cases and of punctured wounds of the orbit in two cases.

I will pass over most of the more important subjective and objective symptoms, confining myself mainly to the question of treatment. In discussing this, however, it is necessary to pick out one or two of the more important symptoms so as to obtain some record as to the way they are affected in the various methods adopted. Proptosis is usually the first

[2] Transactions of the Royal Medical and Chirurgical Society, vol. lviii.

symptom to appear and many patients come for advice complaining of nothing more than some prominence of the eyeball, combined usually "with noises in the head," compared to the whirring of machinery, the buzzing of bees, or the thud of a steam-hammer. This proptosis generally appears during the second month, though occasionally its onset is much earlier, and in one case was not noticed till nearly a year after the accident. The eyeball is dislocated downwards and outwards, the main pulsating tumour occupying the upper and inner angle of the orbit. All the main symptoms were diminished by compression of the common carotid of the affected side and the symptoms steadily increased in most cases when no treatment was adopted, the globe being pushed further and further out of its socket, even on to the cheek. The cornea sloughed and the eye was completely destroyed. Repeated and profuse hæmorrhage will ultimately lead in these cases to a fatal result. In some few cases the symptoms remained quiescent, whilst in others expectant treatment resulted in marked improvement. The treatment adopted in these 20 cases was as follows. 1. In 12 cases the common carotid was tied with a primary ligature in six cases and with a secondary ligature after failure of other methods in six cases. The result was great improvement in all. 2. In four cases no treatment was adopted beyond rest, &c., combined with the administration of sedatives and of iodide of potassium in large doses, with the result that two improved in their general condition, one remained *in statu quo*, and the fourth case was unable to be traced. 3. In ten cases digital or instrumental pressure on the common carotid was tried first, with the result that three showed marked improvement, one remained *in statu quo*, whilst in the other cases, either on account of the pain produced or from failure to obtain any satisfactory result, the common carotid was ligatured, in each case with an immediate successful result. The actual method of compression adopted varied in the different cases; in some a continuous mechanical pressure was used by means of an instrument, whilst in others an intermittent digital method was tried. In one case continuous pressure was adopted for one month and in another intermittent pressure was used for ten weeks, and in each case the result was a complete failure to produce a successful result. In the 12 recorded cases where the common carotid was ligatured the result obtained was an immediate success, the main aneurysmal symptoms rapidly disappeared, the pulsation ceased, the proptosis diminished, and the patients were later able to return to their former occupation. In one case only, however, was there a complete

return to the normal, for in all the others some permanent defect in vision remained, due mainly to the previous stretching of the parts, and in two cases the operation was not carried out sufficiently early to prevent some sloughing of the cornea. The noises in the ear, also, though much diminished, occasionally remained permanently.

The conclusions to be drawn from these cases, according to the method adopted, are as follows: 1. By means of rest, &c., the progress of the disease may be arrested and marked improvement may result. 2. Compression, digital or instrumental, intermittent or continuous, is painful, tedious, and uncertain as to its result. 3. Ligature of the common carotid will almost certainly greatly improve the local conditions and will probably effect a cure, and the earlier the ligation is performed the greater is the chance of reducing to a minimum the subsequent corneal sloughing, &c. No cerebral disturbances need be feared as a result of tying the common carotid unless as a result of the original injury some laceration of the same side of the brain be present. In such cases it is best to accustom the brain to its future condition by some "preparatory" form of treatment as could be brought about by occasional intermittent pressure before the more radical treatment.

Post-mortem evidence of the lesion present.—Three of Rivington's cases showed a direct communication between the internal carotid artery and the cavernous sinus. In the 20 subsequent cases which I have collected an examination was only held in one, and in this case the artery communicated with the sinus by two rounded holes through which the arterial blood was pumped into the cavernous sinus. A united fracture of the petrous bone ran inwards towards the sinus.

The basic fracture peculiar to these cases.—I have previously pointed out that in basic fractures, wherever the injury may be received, the body of the sphenoid, forming one of the weakest of the basic areas, is especially liable to injury, and I shall show later two specimens where the internal carotid artery was lacerated in this region. The exact line of the fracture is not stated in the majority of the recorded cases, but putting all the facts together it appears probable that when orbital aneurysm results from a blow on the head the most common fracture producing such a result will be one which travels along the anterior border of the petrous bone to the cavernous sinus, injuring the internal carotid artery as it runs forwards along the outer part of the cavernous sinus.

2. *Internal Hæmorrhages.*

According to the relation of the extravasated blood to the meninges and cerebrum these hæmorrhages may be classified in the following groups: (1) extradural; (2) intradural; (3) cortical; and (4) intraventricular.

Extradural hæmorrhage may be arterial or venous, in the former case the blood being derived almost exclusively from the middle meningeal artery. A purely venous extradural hæmorrhage is occasionally seen in connexion with fractures of the posterior fossa, in those rare cases where the lateral sinus is mainly torn on its outer aspect. The extravasated blood is unable to find any means of escape owing to the dense mass of muscles attached to the occipital bone, and as the dura mater is in this region but loosely applied an extensive extradural clot results. The middle meningeal artery and its numerous branches are necessarily very liable to injury in all fractures involving the temporal region and the middle fossa of the skull, the anterior branch of the artery being especially liable to such injury. The anterior branch may be torn in the temporal region and so secured with comparative ease, whilst in other cases the main artery, or one of its branches, may be cut across low down in the basic fossa by a fracture travelling obliquely across the middle fossa. In these cases no ordinary trephination will suffice to reach the bleeding point. In the great majority of cases the vessel is lacerated on the same side as that on which the injury was received, but occasionally the hæmorrhage occurs on the opposite side and is not necessarily associated with a corresponding basic fracture. Such cases are almost confined to late life, when the arteries are atheromatous. English records three cases, all of which were fatal, where both vessels were injured.

Uncomplicated middle meningeal hæmorrhage is rare and the coexistence of a fracture of base or vault, with the addition of brain laceration, renders a correct and early diagnosis exceedingly difficult and often impossible. I have previously pointed out that the pressure effects depend to a large extent on the presence or absence of a safety valve such as is afforded by the existence of a comminuted fracture of the tegmen tympani, allowing of the escape of blood into the middle ear and so externally through the gaps of a lacerated drum (Fig. 10). A safety valve is also provided in those cases where the meningeal hæmorrhage is associated with a comminuted fracture of the temporal fossa walls, in which case the extravasated blood escapes into the temporal muscle, leading to swelling, ecchymosis, and even pulsation in that region

(Fig. 13). Compression of this temporal hæmatoma results in an increase of the general compression symptoms and also occasionally leads to twitchings, &c., of the muscles of the opposite side of the face and body. The amount of blood extravasated varied in the different cases from one drachm to several ounces, the largest coagulum being four and a half inches in diameter and one inch thick. The clot, partly fluid and partly jelly-like, is easily removed, except on the dural aspect where it adheres, the removal of the clot leaving the dura mater rough, fibrinous, and discoloured. The dura mater is firmly adherent along the upper border of the temporal bone and also along the lesser wing of the sphenoid, whilst the intervening portion is but loosely attached to the underlying bone of the middle fossa. The blood first poured out has a comparatively easy task in lifting up this loosely applied dura mater, but though exercising an ever greater mechanical pressure as the size of the clot increases, yet it has now to meet insurmountable difficulties in front, behind, and on the inner side, with the result that the blood forces its way upwards towards the convexity of the brain.

Krönlein divides middle meningeal clots into three main varieties, according to the area of brain mainly compressed: (1) temporo-parietal (the commonest); (2) parieto-occipital (rare); and (3) parieto-frontal (very rare). The great difficulties which arise in the diagnosis of an extradural hæmorrhage are evidenced by the following group of cases. An extradural hæmorrhage, of a greater or lesser degree, was found in 30 per cent. of all cases where a post-mortem examination was held and a previous diagnosis was made in 9 per cent. of cases only. The question at once arises as to what are the most important and most suggestive features indicating the probable presence of such a hæmorrhage for the typical symptoms are generally obscured by the presence of other lesions. From my own experience I should put the suggestive symptoms in the following order, according to the frequency of occurrence: (1) the presence of a temporal hæmatoma; (2) a copious and continued hæmorrhage from the ear; (3) twitchings of the facial muscle of the opposite side and twitchings or spasticity of the muscles of the opposite arm and leg; (4) a brief lucid interval between the stages of concussion and collapse; and (5) some alteration in the pupil of the affected side, more commonly a "constricted" pupil. The dilated or Hutchinson pupil is, according to my experience, but rarely seen, except in the later stages, when its diagnostic value is of less importance as the "crisis" is passed. Hutchinson concluded that the dilated pupil was the result of direct pressure on the third nerve. Against this view two points may be urged: (1) no other symptoms pointing to

pressure on the nerve are present nor are any of the accompanying nerves involved; and (2) the hæmorrhage would need to overcome almost insurmountable difficulties in order to lift up the dura mater as far as the outer wall of the cavernous sinus. Pagenstecher and H. P. Dean have also shown that localised compression on the cortical motor areas affects the pupil of the same side, producing first constriction and later dilatation of the pupil. The pupil changes are therefore probably cortical in origin. The temperature, the respiration, and the pulse are so variable that no dependence can be placed on them.

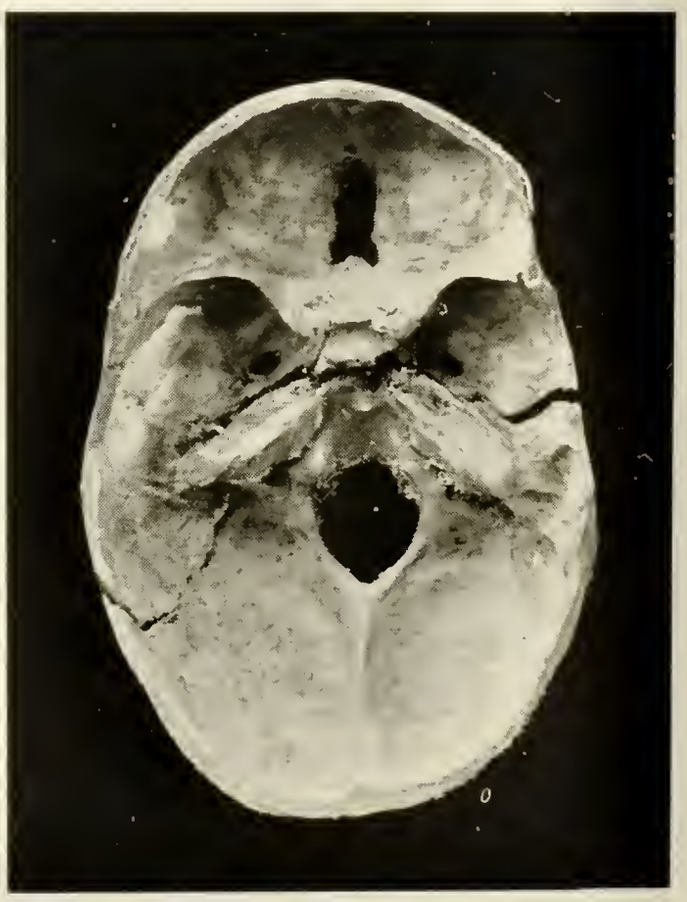
In trephining it is necessary to operate at the earliest possible moment and in doubtful cases it is better to operate and to find no hæmorrhage than to defer any radical treatment till too late. It is frequently necessary to chip away the bone towards the base, as the artery is often torn, low down in the base, soon after the bifurcation. If all local measures fail to reach the bleeding spot it will be necessary to tie the common or external carotid artery. Liddell, in the *American Journal of the Medical Sciences*, reported two cases where it became necessary to ligature the common carotid artery, the result being in each case successful. It is, however, probably advisable to tie the external carotid in preference to the common carotid artery, the brain being already compressed and probably lacerated and any further interference with its blood-supplying may lead to serious early and remote cerebral changes.

Prognosis.—This is best illustrated by the 257 cases collected by Weismann. In 110 cases operated on the mortality was 27 per cent. In 147 cases not operated on the mortality was 88 per cent. In estimating the value of early surgical treatment in connexion with these statistics it must be borne in mind that the majority of the fatal cases not operated on were probably inevitably fatal, owing to the presence of other lesions of the skull and the brain. Weismann's cases would have been of greater value if they were grouped after Jacobsen's plan: (1) hopeful cases; (2) less hopeful cases; and (3) cases hopeless from the first.

Injury to the internal carotid artery.—This vessel, during its passage forwards and inwards towards the posterior clinoid process, lies behind the ordinary fracture which passes across the middle fossa, but as it passes forwards in the outer wall of the cavernous sinus towards the anterior clinoid process it crosses the line of fracture from one middle fossa to the other. Although it is loosely imbedded in the sinus wall, protected by nerves on its outer side, and situated well above the basic level, yet it is in this part of its course that it is liable to

injury. I shall show you two such cases, one in which both vessels were completely torn across, a fracture passing from one middle fossa to the other with such marked separation of the anterior and posterior segments of the skull that the two parts were freely movable, one against the other (Fig. 11), whilst in the second case a fracture, originating in the left frontal region, passed backwards across the anterior fossa to the sphenoidal region widely separating the left middle fossa

FIG. 11.



An extensive fracture passing through the sphenoid from one middle fossa to the other with laceration of both internal carotid arteries.

from the body of the sphenoid, lacerating the left cavernous sinus and partially tearing through the corresponding carotid artery (Fig. 12). Death in each case was nearly instantaneous, blood gushing from the mouth and the nose. The injury to the artery may be in other cases so slight that a fistulous communication between the artery and sinus results, with the formation of an orbital aneurysm.

Hæmorrhage from venous sinuses.—Two of the cerebral venous sinuses are placed in such a situation that they incur

special liability to injury. These are the cavernous and the lateral sinuses. Enough has been said with regard to hæmorrhage from the former sinus under the headings of nasal, naso-pharyngeal, and orbital hæmorrhage. The lateral sinus may be laid open in any part of its course, the rent being almost always more extensive along the inner and unprotected wall. The resultant extravasation is mainly intradural, the blood gravitating first into the cerebellar fossæ and only secondarily tending to be diffused over the cerebral cortex.

FIG. 12.

A severe fracture of the left anterior fossa, extending backwards to the middle fossa, tearing open the left cavernous sinus and lacerating the left internal carotid artery.

The torcula generally escapes injury as the fracture avoids the occipital protuberance and chooses the weaker lateral region. The most common site of laceration is, however, at the angle of junction between the sigmoid and the lateral sinus. This part corresponds to the occipito-mastoid suture and separation along the line of this suture is a frequent sequel to blows received in that region (Fig. 2). The degree of separation is frequently great and this, combined with the dural transmission of the forces, suffices extensively to lacerate the inner

unsupported sinus wall. In the specimen shown (Fig. 13) both sinuses were torn in this situation and besides an extensive intradural extravasation the extradural hæmorrhage was so marked that the dura mater lining the posterior fossa was stripped up throughout its whole extent, from the internal occipital protuberance to the margin of the foramen magnum. The special symptoms produced by such extensive intradural venous hæmorrhage are impossible to specify, for the injury in

FIG. 13.

Probes are passed into the two venous sinuses at the seat of laceration. Comminuted fracture of the left tegmen tympani, etc.

most cases is of such a nature that the patient dies almost at once or is when seen in a moribund condition, owing mainly to the early application of the vital medullary centres.

Hæmorrhage into the subarachnoid and subpial regions.—In most cases of severe head injury hæmorrhage occurs in these situations and on reflection of the dura mater a blood film is seen, of variable extent and thickness, extending over the cerebral cortex and dipping down into the major and minor

sulci. When localised it is most commonly situated over that part of the brain which lies directly opposite to that part of the skull at which the injury was received and it is produced by the impact of the brain against its bony barrier. When situated over a motor area the symptoms vary according to the position and extent of the hæmorrhage. When slight in extent symptoms of irritation result, with severe localised headache, contracted pupils, and some spasticity of the regions supplied by the affected area. When more severe localised twitchings may result from the irritation of a particular motor area, the muscular movements involving one part after another as the effused blood becomes more diffused. The twitchings may subsequently give way to a spastic condition, but actual paralysis is rare, such a condition generally indicating some cortical laceration.

Intraventricular hæmorrhage.—Hæmorrhage into the lateral, third, and fourth ventricles is only seen in the most severe cases, and death generally results early. Symptoms resembling those seen in apoplexy are said to result.

The Escape of Cerebro-spinal Fluid from the Nose and Ear.

This symptom occupies in text-books so prominent a position that one is led to the belief that a discharge of cerebro-spinal fluid from the ear or from the nose is of common occurrence. Such, however, is not my experience The escape of a clear or slightly blood-stained fluid does not necessarily imply that the fluid is cerebro-spinal in nature, for it has been proved on numerous occasions that watery fluid may escape in considerable quantities from the ear or from the nose without the existence of any basic fracture, the fluid in such cases being derived either from the membranous labyrinth (the liquor Cotunnii) or from the mucous membrane of the ear or nose as a result of the great vaso-motor dilatation of the tympanic and nasal vessels. If the fluid be cerebro-spinal in nature the natural inference is that the subarachnoid space is opened up to the exterior, either directly or indirectly along the course of a cranial nerve. A chemical examination of the fluid may help in the diagnosis since the two fluids differ appreciably in composition.

1. *Cerebro-spinal fluid* is rich in chlorides, contains little or no albumin, and a trace of a reducing substance allied to pyrocatechin.

2. *Fluid derived from other sources* contains chlorides, a fair amount of albumin, and no reducing substance.

Certain factors, however, enter into the case which neutralise the advantages of a chemical analysis, for even though the fluid be cerebro-spinal in nature it is frequently mixed with blood, with the consequent addition of albumin, whilst if the fluid be profuse in amount the first part only contains any reducing substance, the fluid soon becoming a mere serous exudation. I have several times been able to collect a clear fluid from the ear, drop by drop, in a test-tube, and I have generally failed to demonstrate the presence of this reducing agent, though the post-mortem examination evidenced later that the fluid was derived from the sub-arachnoid space. The fluid is almost certainly cerebro-spinal in nature: (1) if the discharge begins within 24 hours of the accident; (2) if the discharge is colourless and profuse; and (3) if the discharge continues for some days. The time of onset, the duration, and the quantity are all, however, subject to great variation. 1. The time of onset is usually early, but cases are recorded where the discharge began weeks after the accident. 2. The duration may be from a few hours to many weeks. Sir W. Savory recorded a case where fluid escaped for one month from both ears. 3. The quantity varies from a few ounces to several pints. Sir William MacCormac described a case in which three pints escaped in five hours.

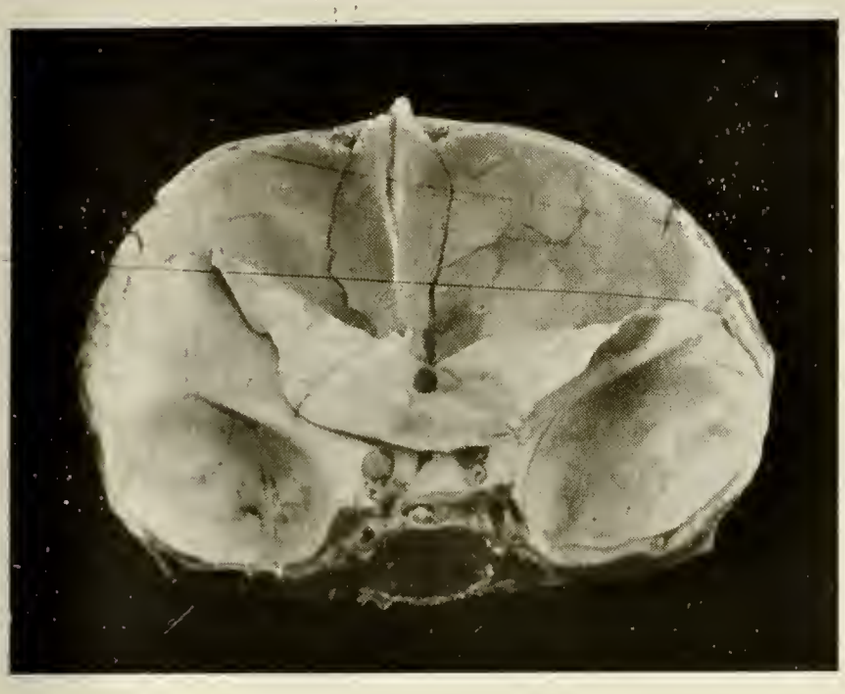
This occasional great loss of fluid is due to the fact that as the cerebro-spinal fluid escapes the pressure in the sub-arachnoid space is practically reduced to zero, and since the cerebro-spinal tension and the intracranial venous pressure are equal fluid continually passes from the veins into the subarachnoid space as long as the cerebro-spinal fluid escapes.

Cerebro-spinal fluid from the nose.—Blandin, of the Hôtel Dieu, first drew attention to this symptom in 1840. The gross lesion most commonly present is a comminuted fracture of the cribriform plate of the ethmoid, with laceration of the overlying dura mater and arachnoid (Fig. 14). More rarely the fluid may be derived from the cisterna basalis, the fracture involving the sphenoidal body with laceration of the overlying membranes and with comminution of both roof and floor of the sinus. Goucard and Malgaigne both describe cases where, as the result of a severe fracture of the petrous bone without injury to the membrana tympani, the fluid escaped forwards into the naso-pharynx along the Eustachian tube.

Cerebro-spinal fluid from the ear.—This was first recorded by van der Wiel in 1727, and more completely by M. Langier in 1839. The source of the fluid is usually stated to be as follows: "The fracture involves the petrous bone in such a way that the arachnoid and dural sheaths round the seventh

and eighth pairs of nerves are torn, allowing the cerebro-spinal fluid to escape into the middle ear, and so externally through a torn membrana tympani." The great majority of fractures which involve the petrous bone pass in front of the genu of the seventh nerve and in front of the eighth nerve and internal ear. Some further lesion is therefore necessary, and it will be found that two main varieties of fracture may lead to aural discharge of cerebro-spinal fluid. 1. A fracture of the middle fossa, with such comminution of the petrous bone that the subarachnoid space is opened up, either directly by laceration

FIG. 14.



Fracture of the anterior fossa with laceration of the dura mater overlying the cribriform plate. Both frontal sinuses are involved.

of the dura mater and arachnoid overlying the tegmen tympani (Fig. 10) or indirectly by involvement of the membranous prolongations along the seventh and eighth nerves (Fig. 4). 2. A fracture of the posterior fossa which, on reaching the jugular foramen, cuts across the petrous bone, traversing the vestibule, and severing the facial nerve in the region of the genu (Fig. 5). The external signs of injury and the condition of the facial muscles will generally suffice to make clear which variety of fracture is present.

Prognosis and treatment.—The discharge of cerebro-spinal fluid does not materially influence the prognosis. Large quantities of fluid may be lost without any striking effect,

and though the existence of such a discharge necessarily indicates the opening-up of the subarachnoid space to the exterior, yet the danger of infection does not appear to be increased proportionately, the outflow tending, no doubt, to wash away any micro-organisms present in the external meatus. Battle records 36 cases with a mortality of 25 per cent., and another writer 11 cases with a mortality of 27 per cent., the general mortality in all cases of fractured base being estimated at 32 per cent. As regards the special treatment in these cases it is most inadvisable to syringe out the ear, as by such means a stream of fluid is driven into the middle ear and even further if the tegmen, for instance, be extensively comminuted, the danger of infection being proportionately increased. The meatus should be merely swabbed out with some strong antiseptic, a little iodoform powder lightly blown in, and absorbent protective dressings applied, the whole being maintained accurately in position by firm and efficient bandaging. Frequent changing of the dressings may be necessary when the discharge is profuse. The patient must be carefully watched in case attempts may be made to interfere with the bandages and such action prevented by tying down the hands to the side. It is important to bear this in mind as such attempts are frequently made. When the fluid escapes from the nose or into the naso-pharynx little can be done to lessen the risk of infection, and as a result meningitis is most commonly secondary to a fracture of the anterior fossa.

LECTURE III.

Delivered on March 4th.

Implication of Nerves in Basic Fractures.

Mr. President and Gentlemen,

I have already shown that fractures of the base tend to involve many of the basic foramina. The nerves, however, which make their exit through these foramina are protected by strong dural sheaths, which fact accounts for their frequent escape from injury. Nevertheless, many of the nerves are liable to injury and I propose, therefore, to discuss briefly each nerve in turn with respect to this special liability, indicating the particular fracture present, with the result and prognosis.

1. *The olfactory nerve.*—The great majority of fractures involving the anterior fossa pass through the weak cribriform plate of the ethmoid and necessarily injure the fine filaments of the olfactory bulb (Fig. 14). The bulb itself may be lacerated, with or without injury to the neighbouring parts of the under aspect of the frontal lobes. Such an injury may also be produced as the result of a blow over the occipital region, an exemplification of laceration by contrecoup. Sir Prescott Hewett considered that loss of smell is most frequently the result of blows on the back of the head. My experience, however, goes to prove that anosmia, unilateral or bilateral, transient or permanent, results almost invariably from direct fractures involving the cribriform plate. The actual proof of an immediate loss of smell is difficult to arrive at since the nares are usually blocked by blood clot, &c., but a careful examination at all stages of the illness and of the convalescence shows that temporary loss of smell is common, whilst permanent anosmia is rare.

2. *The optic nerves.*—Cases have from time to time been recorded of complete and sudden blindness of one eye following a severe head injury, and at first sight this result is not easily explained, since the great majority of fractures in the vicinity of the optic foramina tend to pass to or from the cribriform plate between the two foramina or more externally through

the sphenoidal fissure. Small fissured fractures not infrequently radiate to the optic foramina, but they are usually of so slight a nature as to be incapable of causing any gross lesion of this nerve. No doubt hæmorrhage into the sheath of the optic nerve is responsible for a certain small proportion of these cases but taking into consideration the very common existence of a fracture through the base of the anterior clinoid process, and the frequent dislocation of the fragment, it is highly probable that the loss of vision is secondary to a fracture tearing off the corresponding anterior clinoid process, the fragment compressing or tearing the optic nerve beneath (Fig. 3, A and D). These cases are so rare that I am only able to put this forward as a suggestion.

3. *The nerves passing along the cavernous sinus and through the sphenoidal fissure. The ophthalmic division of the fifth nerve.*—The main trunk of this nerve as it runs along the outer wall of the cavernous sinus is never subject to such injury as to result in anæsthesia of the parts supplied by all three of its terminal divisions. The extravasation of blood into the peri-orbital tissues may, however, result in a complete loss of function of one or more of its branches and cases are recorded where there has been complete anæsthesia of both cornea and conjunctiva with subsequent ulceration and sloughing. The nasal branch may alone be implicated as the result of a fracture involving the cribriform plate and the supra-orbital and supratrochlear divisions may be damaged by a fracture of the vertical plate of the frontal bone. When the anæsthesia is the result of pressure from extravasated blood the prognosis is favourable, but when a nerve is involved in the fracture the anæsthesia is generally permanent. The *third* nerve is similarly liable to injury in its orbital course, the only peculiarity being that some of its branches are picked out and the others left uninjured. The *fourth* nerve is occasionally involved but always in conjunction with other nerves.

The second and third divisions of the fifth nerve.—The foramen rotundum and the foramen ovale lie anterior to the petro-sphenoidal suture, along which suture most middle fossa fractures pass (Figs. 11 and 17). These two nerves consequently escape direct injury, though they may rarely be involved, together with the first division of the fifth nerve, through implication of the Gasserian ganglion. In 1852 H. Lee[1] reported a case where seven weeks after the injury the following symptoms were present: anæsthesia of the left face and forehead, anæsthesia of the anterior two-thirds of the

[1] Medical Times and Gazette.

tongue on the left side, anæsthesia of the left nostril, weak left masticatory muscles, and opaque left cornea. The fracture peculiar to such cases is one in which the terminal part of the apex of the petrous bone is broken off in an antero-posterior direction, the fracture traversing the cavum Meckelii. These fractures are rare and I have only seen two cases.

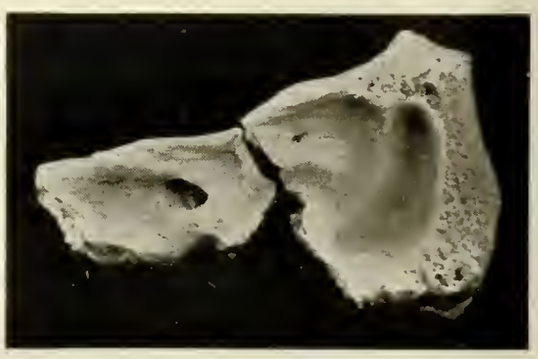
The sixth nerve may be either compressed, in conjunction with other nerves, by extravasated blood during its passage forwards from the cavernous sinus or it may be the only nerve paralysed, with consequent abductor paralysis of the eyeball. Such cases are not uncommon and this isolated paralysis of the sixth nerve results from the implication of the nerve as it laterally grooves the dorsum ephippii, which process is occasionally torn across in a fracture passing from one middle fossa to the other (Fig. 11). The paralysis is generally unilateral, the fracture crossing the dorsum ephippii in an oblique direction.

The seventh and eighth nerves.—There can be no doubt that the seventh nerve is more commonly involved than any other nerve in basic fractures owing mainly to its complicated intrapetrous course. Köhler records 22 cases in 48 cases of fractured base; Bidwell records 15 cases in 106 cases of fractured base: and Battle records 15 cases in 168 cases of fractured base. My own experience leads me to the view that Köhler's statistics are the more accurate, as 24 cases of facial paralysis, partial or complete, were seen in 60 cases of fractured base, 12 in 32 cases that recovered and 12 in 28 fatal cases.

The question of facial nerve implication is so bound up with auditory symptoms that the two subjects must be treated together and classified into two main groups: (1) early and partial paralysis of the facial nerve, accompanied by a variable degree of deafness; and (2) early and complete paralysis of the facial nerve, accompanied by complete deafness. In the first group are iucluded the great majority of middle fossa fractures, such fractures tending to involve the auditory region, and evidenced, amongst other symptoms, by hæmorrhage from the external auditory meatus. A greater or lesser degree of facial paralysis is usually present, not always obvious at first sight, but requiring a careful scrutiny and comparison of the two sets of facial muscles. The fracture passes inwards along the roof of the external auditory meatus towards the junction of the anterior and internal walls of the middle ear, the membrane undergoing a variable degree of destruction with or without dislocation of the ossicles. The fracture then passes inwards to the region of the petro-sphenoidal suture in such a manner that

the geniculate ganglion of the facial nerve is exposed and laid bare on the anterior aspect of the posterior fragment (Fig. 4). The main trunk of the nerve consequently escapes injury except in the region of the genu, where it may be compressed by blood clot sufficing to produce a partial temporary loss of function. In most cases the clot is entirely absorbed and a complete recovery can be expected. The degree of deafness varies according to the extent of damage done to the membrane and ossicles, such being most extensive when the injury results from a direct blow over the auricular region. In many cases, however, the recovery is complete. In the second class of cases the fracture is quite different. Usually the result of a blow on the occipital region, the fracture traverses the thin cerebellar fossa to the outer margin of the jugular foramen. Thence the fracture cuts

FIG. 15.

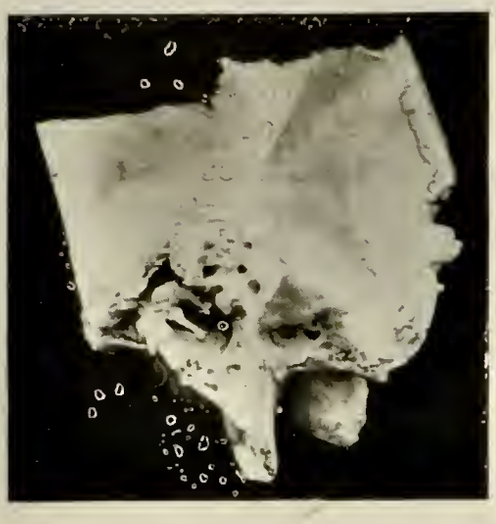


The posterior aspect of the right petrous bone, showing a fracture running in the antero-posterior direction.

across the petrous bone, external to the internal auditory meatus, and terminates usually by comminuting the tegmen tympani (Fig. 5). It is in this transpetrous course of the fracture that the irreparable damage is done, for not only is the facial nerve cut clean across in the region of the genu but the auditory apparatus is also severed into two parts. The exact line of the fracture is shown in Fig. 15. The outer fragment is the more interesting and the next two figures explain in detail the parts involved. Fig. 16 shows the open mouths of the semicircular canals, the outer parts of the fenestra ovalis, of the promontory and of the fenestra rotunda, and the exposed mastoid antrum, whilst the malleus, the incus, and the membrane are absolutely uninjured. In Fig. 17 the comminution of the tegmen is more extensive but otherwise the parts exposed are similar to the

preceding case. In addition, however, the specimen shows that the fracture involves the petro-sphenoidal suture, passing

FIG. 16.



The outer fragment of the same specimen viewed from the inner side showing the semicircular canals, the facial aqueduct, the ossicles, and the membrana tympani.

posterior to the foramina ovale and spinosum but anterior to the horizontal part of the internal carotid artery. The long probe is passed along the Eustachian tube and the smaller is

FIG. 17.

A more extended fracture of the right petrous bone. This specimen also shows the inner aspect of the outer fragment together with the mastoid antrum, a probe in the facial aqueduct, the ossicles, the membrana tympani, the semicircular canals, the internal carotid artery, a probe in the Eustachian canal, and the foramen ovale.

insinuated into the horizontal part of the aqueductus Fallopii. In these cases, therefore, though the facial paralysis and deafness are both complete and permanent, there is no external auditory hæmorrhage.

Two points further need reference, the first being the rare variety of fracture described by Léon Boullett in 1878, under the title of "fracture of the mastoid portion of the temporal bone" secondary to direct blows on the mastoid process, with a consequent separation of that process from the rest of the skull, the facial nerve in the descending part of the aqueduct usually being injured. Lastly, *late* paralysis of the nerve is occasionally seen after the lapse of a few days, secondary either to a septic infection with an ascending neuritis or to the changes which take place in the blood clot around the genu of the nerve. The diagnosis between these two conditions is simplified by the presence or absence of pyrexia, rigors, vomiting, &c.

The ninth, tenth, and eleventh nerves.—The jugular foramen lies in the path of more than one line of basic fracture (Fig. 2). The nerves, however, are well protected and usually escape injury. Cases are now and again seen in which the result is less favourable, the following being a brief abstract of such a case. The patient was admitted with a fracture of the posterior part of the parietal bone and for four days, beyond restlessness, no special symptoms were evident. On the fifth day there was a sudden attack of severe dyspnœa with diaphragmatic respiration only. The conditions improved till the sixth day, when he was again seized with another attack of acute dyspnœa, accompanied by dysphagia, during which death occurred. The examination showed a fissured fracture extending into the jugular foramen which was occupied by a blood clot. A similar case was recorded in The Lancet in 1853, in which the patient, who was admitted with a fractured base, was doing well till the tenth day, when, on getting up from his bed, he was seized with rigors, vomiting, dysphagia, and dyspnœa, dying shortly afterwards. A fracture was found practically dividing the skull into two halves and involving the jugular foramen. Displacement of the two parts had taken place with compression of the nerves. Delfau and Günther narrate a case where death resulted from compression of the vagus. The only case that has come under my own personal notice in which the structures passing through the jugular foramen were directly involved was that of a man who committed suicide by firing a bullet through the roof of the mouth. The bullet, impinging against the tympanic plate, was divided into two parts, the outer fracturing the floor of the external auditory meatus, the inner lodging in the jugular fossa,

lacerating the lateral sinus and tearing the nerves as they made their exit through the jugular foramen. Death was instantaneous.

The twelfth nerve.—I have no personal experience of fractures involving the foramen through which this nerve passes and on comparing the position of the anterior condyloid foramen with the definite paths of basic features to which I alluded in my first lecture any implication of this foramen appears to be highly improbable (Fig. 3).

The Temperature in its relation to Head Injuries.

For some considerable period of time it has been a well-recognised fact that injuries of the head result in definite temperature changes. Broca, Battle, Guyon, and others have endeavoured to deduce facts from an examination of such cases but no definite result was arrived at beyond the fact that there was some ratio between the change in temperature and the co-existent lesion. In all those cases which were seen by me and in which a post-mortem examination was subsequently made a comparison was instituted between the temperature changes during life and the lesions found after death. The conclusions arrived at were as follows: 1. For a short and variable period of time after the receipt of the injury the temperature is always subnormal, sometimes so low that no thermometric calculation can be made. This is the period of shock which is in all severe cases very pronounced. 2. If the patient lives long enough reaction soon takes place and the temperature rises. 3. This rise of temperature is in fatal cases rapid and progressive. In one case the temperature rose 6° F. in seven hours and in another 8° in four hours. 4. In some cases the temperature rises to a more moderate height, rarely more than 102°, and then "marks time." This period marks the crisis of the case, for a subsequent rise or fall almost invariable indicates a fatal or favourable result. I should explain, perhaps, that I am excluding for the present any alteration in temperature due to the onset of septic or pulmonary complications. 5. The rise of temperature is not dependent on any special bony lesion, since the same changes are seen in fractures of the vault and in fractures of the base, anterior, middle, or posterior fossa. 6. Laceration or contusion of the brain is generally present. The almost constant presence of some injury to the cerebral substance gave origin to the old theory that the temperature changes are secondary to brain laceration. Against this view, however, several facts can be brought forward: (*a*) Laceration is not always present even when the temperature runs high.

In two cases, for instance, the temperature rose to 106° and yet there was no sign of contusion or laceration. (*b*) Laceration, even when extensive, is not necessarily followed by any elevation of temperature. (*c*) The elevation of temperature is absolutely independent of laceration of any particular area. Richet[2] arrives at the conclusion that two hypotheses present themselves, accounting for the elevation of temperature, between which it is difficult to decide. Either there are certain temperature regulators in the encephalon which when excited become stimulated in function, or else the cortical injury acts in a sort of reflex manner on the regulatory centres situated in the pons or in the bulb. Attention is also drawn to the experiments of Lorin and van Benedin who, after excising the two cerebral hemispheres of a pigeon, showed that the heat regulatory power was preserved unaltered, proving that the corpus striatum is probably not the head office of the heat-regulating apparatus. Till, therefore, more evidence comes to hand with regard to heat regulation in general and the effects produced by injury, one cannot go beyond the broad statement that practically all severe cases of head injury show definite thermometric changes, the elevation of temperature not resulting from any special lesion of bone or of cortex but being probably due to the influence of the injury on the heat-regulating centres in the region of the pons or bulb, the temperature progressively rising as the cardiac and respiratory centres fail.

The next point to consider is the value of the temperature chart with regard to the prognosis of the case. All cases of fractured skull can be divided into three main groups, according to the thermometric changes, excluding any alterations secondary to septic, pulmonary, or other complications.

Group 1.—The temperature, at first subnormal, undergoes a steady and progressive rise. The prognosis is most unfavourable.

Group 2.—The temperature, at first subnormal, rises gradually to 101° to 102° and then for a short time remains steady. This hesitation marks the crisis of the case, a further rise indicating a probable fatal result, whilst a fall in temperature offers hope of future recovery.

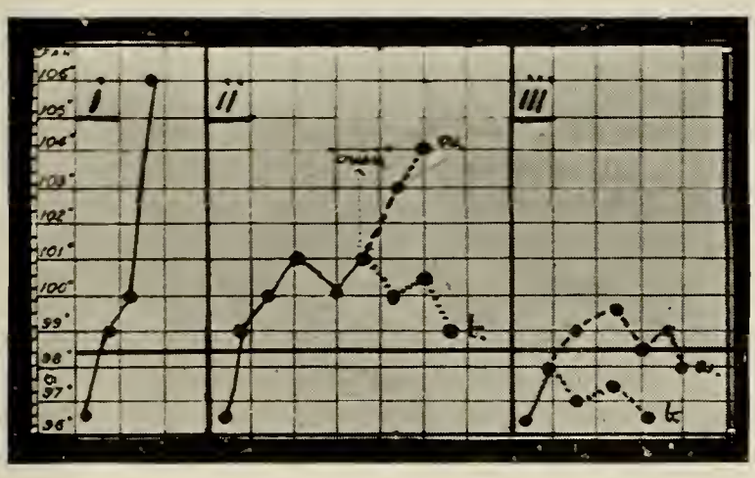
Group 3.—The temperature, at first subnormal, remains at that level, or rises to about normal without any subsequent further elevation. In the former case the patient, owing to the severity of the injury, never recovered from the stage of collapse, whilst in the latter case the injury was of so comparatively slight a nature that the stage of shock, being

[2] Dictionnaire de Physiologie.

neither prolonged nor deep, was not subsequently followed by the stage of reaction.

These three groups of thermometric changes are so constant that I regard the temperature chart as the surest and as an almost infallible guide to the prognosis of the case. With regard to any alteration of temperature on the two sides of the body, in spite of an occasional relative increase of temperature on the side opposite to the injury, I have not been able to satisfy myself that such changes are sufficiently constant as to be of any diagnostic value.

Fig. 18.



The temperature charts of the three typical groups.

Changes in the Fundus Oculi.

In 1887 Edmunds and Lawford published a paper in which attention was first drawn to this subject and statistics were given stating that optic neuritis was present in 12 out of 24 cases of head injury. A subacute basal meningitis was considered to be the cause of this neuritis. In 1890 Battle gave details of 17 cases, in five of which optic neuritis was diagnosed, and he suggested as a cause laceration of the under aspect of the frontal lobes. On reading these statements two questions must arise. Firstly, Are the explanations offered satisfactory; and secondly, Why should optic neuritis develop in cases of head injury uncomplicated by secondary septic infection? These points induced me to examine all cases that came under my notice, an examination of the fundus being carried out at all stages of the illness, with the result that two distinct varieties of fundus change were seen: (1) a disc with normal outline, the fundus, however, showing marked engorgement of the retinal veins, with small arteries; and (2) a fundus

presenting all the signs of true optic neuritis. This latter variety was seen only in those cases which subsequently succumbed from septic meningeal infection, whilst the venous retinal engorgement was of common occurrence.

Now, one of the most striking features in the post-mortem examination of cases of head injury is the great engorgement of the superficial cerebral veins and this venous congestion is seen equally in fractures of the vault and in fractures of the base, with or without brain laceration, and also in cases of mere concussion only. The retinal engorgement is early in onset, being frequently seen within a few hours of the injury and long before any true optic neuritis could develop. It is most marked when the injury involves the anterior segment of the skull and it is not necessarily accompanied by laceration of the under surface of the frontal lobes. It is, however, most common when a fracture traverses the sphenoidal fissure or involves the cavernous sinus. As the patient improves, so also does the congestion diminish in direct ratio, any later development of true optic neuritis indicating the onset of an acute or of a subacute meningitis. I regard, therefore, this retinal venous engorgement as part and parcel of the general cerebral venous stasis, the condition being of no great clinical value with regard to the presence or absence of any fracture. Dr. Fleming has recently pointed out that retinal hæmorrhages are almost invariably present in basic fractures and, further, that when the subarachnoid hæmorrhage and venous congestion are unilateral the retinal hæmorrhages are mostly confined to the affected side. The subject of "optic atrophy" has been briefly discussed under "implication of nerves."

Brain Laceration; Concussion and Compression.

Of all complications of fracture of the skull injury to the brain substance must be regarded as of the greatest importance, for on the presence or absence of such a lesion the ultimate result in the majority of cases depends. Some contusion or laceration of the brain is almost invariably present when the injury results from a fall from a height, the lesion varying from a mere bruising of the cerebral cortex, with extravasation of blood and serum into the subarachnoid space and into the meshes of the pia mater, to complete laceration of certain of the cortical areas. No part of the brain can be said to be exempt from such injuries, for though it is far more common to find laceration of the frontal and temporo-sphenoidal poles, yet one not infrequently finds laceration of the Rolandic area, of the occipital poles and cerebellum, and

even of the deep nuclei of the brain. The relative frequency with which these various areas are injured is: (1) the anterior and inferior aspect of the frontal poles; (2) the anterior aspect of the temporo-sphenoidal poles; (3) the Rolandic area: (4) the occipital poles; (5) the cerebellum; and (6) the deep nuclei and the deep cortex.

Laceration may be seen directly beneath the seat of primary osseous injury or on the surface of the brain over an area exactly opposite to that at which the injury was received. Various theories have been brought forward to explain this latter condition. 1. "Their production has been ascribed to the changes in form suffered by the skull in virtue of its elasticity when subjected to violence, which causes distortion of the brain to the point of rupture." 2. "In the displacement of the cerebro-spinal fluid by the consecutive cranial depression and bulging, which is believed to follow a blow on the head, a momentary vacuum is formed at either end of the axis of force and the vessels of the brain and membranes rupture from lack of support." These two theories, both ingenious in their way, are, nevertheless, untenable as they are based on an erroneous supposition. There is no practical evidence whatsoever to prove that the skull as the result of a blow sufficient in force to produce a fracture undergoes any change in shape in virtue of its supposed elasticity, or that "consecutive depression and bulging" is the sequel to the blow inflicted in that region. 3. The contrecoup theory, which states that the injury results from the sudden displacement of the brain against the bony cranium. The theory has been strenuously opposed by many, and notably by Helferich, on the ground that the brain is said to fill completely the bony cranium and, therefore, that any "shaking" of the brain from side to side is impossible. The contrecoup theory affords, however, the most satisfactory explanation of laceration of the brain over an area opposite to the seat of osseous injury, for (1) the frontal and temporo-sphenoidal poles are more subject to injury than any other part of the brain, each pole having peculiar anatomical relations rendering it more liable to injury. The frontal pole is in closer apposition and in closer relation to the bony barrier than any other part of the brain, whilst the temporo-sphenoidal pole projects into the cul-de-sac at the anterior part of the middle fossa which is overhung by the lesser wing of the sphenoid. 2. The laceration is always seen to be situated at the pole directly opposite in the line of the transmitted forces. 3. Bruising, without laceration, is very commonly seen over the area of the brain exactly opposite to the part struck. 4. Laceration on the opposite side is most frequent in those cases

where the injury is produced by falls from a height and it is in such cases that "shaking" of the brain is most likely to occur. The argument also that because the brain completely or almost completely fills the bony cranium "shaking" from side to side cannot occur is in itself fallacious, for the soft cerebral substance can be still more violently and forcibly driven up against the bony barrier as to be locally bruised and torn. That a "shaking" of the brain can occur is evidenced by the following case. A prize-fighter received a blow on the left side of the head and was knocked out. He was admitted into hospital unconscious and died on the third day. The post-mortem examination revealed no fracture of the vault or base but a linear laceration on the mesial aspect of the left hemisphere exactly corresponding in position to the free margin of the falx cerebri, showing that the brain had been so driven up against this firm and resisting barrier as to result in a corresponding laceration of the soft cerebral substance.

The lacerated area is usually cone-shaped, the base corresponding to the cortex, at which situation the brain destruction is most marked, whilst the apex of the cone extends inwards towards the lateral ventricles. In severe cases the anterior or lateral cornua of the lateral ventricle may be involved at the apex of this cone of laceration, and if further softening and fatty degeneration of the damaged cerebral substance subsequently result the horns of the ventricle may bulge, as it were, outwards towards the subarachnoid space, thus assisting in the formation of those cases of traumatic meningocele or meningo-encephalocele which communicate directly with the lateral ventricles. If the degree of laceration be comparatively slight, and if the other injuries are compatible with life, organisation and partial resolution take place with the formation in nearly all cases of adhesions between the cortex and the overlying membranes, with the formation of false membranes, of arachnoid cysts, and of blood cysts, all such lesions being accompanied by a variable degree of fibroid degeneration of the corresponding cortical motor area.

For two main reasons the symptoms resulting from brain laceration are obscure. First, there is almost invariably a coexistent vault or basic fracture; and, secondly, the two areas most commonly involved, the frontal and temporo-sphenoidal poles, do not make evident any injury which they may receive by any peculiar and localising symptoms. When the Rolandic areas are involved irritative and paralytic symptoms may result, but symptoms of irritation are also seen in cases where extensive subarachnoid hæmorrhage is alone present and especially is this the case in children. Convulsions and paralysis, therefore, of the parts depending in function on

the integrity of the cortical motor area are the only definite clinical symptoms indicative of brain laceration. The lesser degrees of laceration may lead to a spastic condition of the parts supplied by the affected area and laceration may be suspected also when the stage of unconsciousness is deep and prolonged. Even in such cases, however, laceration is not necessarily present. The symptoms, therefore, of brain laceration are generally obscured and overshadowed by those which are regarded as typical of the stages of concussion, irritation, and compression. The symptoms of concussion are sometimes regarded as necessarily implying the existence of brain laceration. With this opinion I am not in accord and for two main reasons. First, I have examined several cases where, in spite of concussion symptoms, no macroscopic or microscopic cerebral lesions were found; and, secondly, the rapid recovery of so many cases of concussion must necessarily exclude any such gross cortical lesions, lesions which must require a lengthened period of time before recovery could result. Concussion shows a very close analogy to the condition of shock, with certain superadded cerebral symptoms. Shock is referable to a stimulation of the depressor fibres of the vagus nerve, with consequent vaso-motor inhibition and weakening of the heart's action. The venous dilatation and engorgement, most evident in the splanchnic area, are also marked in the cerebral venous system, the cerebral circulation probably possessing no separate vaso-motor apparatus and therefore passively following the changes in the general circulation. The cerebral venous engorgement is accompanied by a condition of cerebral arterial anæmia which, together with a certain degree of disturbance in function of the cortical cells, results in a temporary cessation of cerebral function, with loss of consciousness, subnormal temperature, feeble rapid pulse, and shallow respiration. (Concussion, first stage.) The injury being comparatively slight the vaso-motor centre soon recovers tone and as the result of the previous inhibition the heart now beats more forcibly and the condition of cerebral arterial anæmia gives place to a state of cerebral arterial hyperæmia, evidenced by violent carotid pulsation, flushed face, elevated temperature, and general cephalalgia. (Concussion, second or reactionary stage.) This stage is usually ushered in by vomiting secondary to the hyperæmic condition of the vomiting centre. The main symptoms of concussion are evoked immediately on the receipt of the injury and they may be either followed by the symptoms peculiar to the reaction stage or they may remain as the prominent feature, only becoming more marked as the vital centres fail, a result common in those more severe cases of

head injury which have an early fatal termination. In some few cases, however, the symptoms merge into those peculiar to compression, with or without an intermediate rational interval.

Some stress should be laid on the fact that in most cases of head injury the typical compression symptoms are conspicuous by their absence. One or more of the symptoms peculiar to compression may be present but the complete clinical picture of cerebral compression is in these cases seldom seen. If the injury be severe, and especially if laceration of the brain be present, the stage of shock is reflexly correspondingly increased, the cerebral arterial anæmia becoming more marked and the venous engorgement increased in direct ratio. The engorgement also of the right side of the heart tends to increase the cerebral venous pressure and since the cerebro-spinal tension and the cerebral venous pressure are equal, the greater the venous pressure the greater is the transudation from the veins into the brain and into the subarachnoid and other cerebro-spinal spaces, the transudation progressing till the two become equal. The cause being removed or the injury being of such a nature as not to exclude an attempt at recovery, the cerebral anæmia becomes less marked, the veins less congested, and transudation back into the veins occurs till a more or less normal condition is again reached. In the most severe cases the venous engorgement remains excessive and the cerebro-spinal tension correspondingly high, the patient passing through the stage of compression to the stage of collapse. An examination of such cases reveals the presence of much free fluid, especially in the cerebellar fossa, and also a general œdematous condition of the brain. Experiments show that from 3 to 6 per cent. of the cranial capacity may be taken up by a foreign body without producing compression symptoms, owing to the escape of a corresponding quantity of cerebro-spinal fluid into the spinal canal. Any increase above this percentage results in compression symptoms, the general increase of cortical pressure leading first to the state of "slow cerebration," a condition characterised by slow speech and by a deadening of the mental powers and finally to a state of stupor and coma.

The special symptoms of compression are, however, probably referable to the effect of the increased pressure on the vital centres in the region of the bulb. That such is the case is suggested by the following facts: (1) the same compression symptoms result wherever the compression force primarily acts; (2) a fracture involving the posterior fossa gives rise to compression symptoms earlier than a fracture in any other more distal region; (3) experimentally an increased pressure in the posterior fossa produces com-

pression symptoms earlier than when the pressure is applied to a more remote situation; (4) a far smaller foreign body kills in the bulbar region than in the cerebral chamber (Leonard Hill); and (5) the pressure effects are in no way due to excitation at the part of the brain chiefly pressed on, for after division of the mesencephalon the pressure was just as active in calling forth the changes in circulation and respiration (Sir Victor Horsley). Leonard Hill came to the conclusion that the main symptoms are produced by anæmia of the bulb and that mechanical pressure was not the main factor. Whether this is the case or not, the main fact stands out clearly that both clinical and experimental evidence strongly suggest that the main compression symptoms are bulbar in origin.

The three stages—concussion, compression, and collapse—may follow one another, the second and the last resulting probably from early stimulation and later paralysis of the nuclei in the floor of the fourth ventricle. As previously stated, however, the severe nature of the lesion in most fatal cases of head injury more or less completely eliminates the transitional stage of typical compression. Death virtually results from paralysis of the respiratory centre, the heart usually beating for some seconds or minutes after all attempts at respiration have ceased. Some years ago I saw a case where, although respiration had ceased, the heart worked well as long as artificial respiration was kept up. This was done by relays of "dressers" for from two to three hours. Our endeavours to resuscitate the patient were then abandoned as hopeless, and in a few minutes the heart ceased also.

Treatment.

Some few points in connexion with the treatment of the results of skull fracture have been alluded to under the different headings and the main points with regard to the general and local treatment now remain.

(*a*) *General treatment.*—In the early stage of shock and collapse the ordinary methods of overcoming that condition must be adopted. The head must be kept low and the heart's action encouraged by the usual remedies. The temperature should be taken every quarter of an hour and as soon as reaction *begins* the head should be elevated and the body depressed, ice applied to the head and warmth to the body, and free venesection performed, the amount of blood to be withdrawn being proportionate to the severity of the injury and the violence of the reaction. These recommendations are advised with the object of allowing some of the extravasated blood and fluid to gravitate into the vertebral canal and with the view of

lessening as far as possible the subsequent intracranial exudation and pressure. Free venesection is especially efficacious in producing this result and the operation is best performed on the external jugular of the affected side. The lower bowel should be emptied early by means of copious enemata, purgatives given, and the bladder periodically attended to.

(*b*) *Local treatment.*—It is now a well-accepted fact that all punctured fractures and all markedly depressed fractures in adults, simple or compound, urgently call for operative interference. A large class of fractures remains, however, in which the question of any depression of the internal table is doubtful and the general tendency in these cases is to adopt an expectant treatment, waiting for any symptoms to develop. Many of these cases during their stay in the hospital do not offer any striking or trustworthy symptoms calling for operative treatment, and yet, if the cases be subsequently followed up, a large percentage develop some of the remoter effects of head injuries, such as chronic headache, irritability, or Jacksonian epilepsy, &c. These secondary results are undoubtedly due in most cases to the irritation produced by bony spicules from a depressed internal table, with blood effusion, with thickening of the meninges, and with the formation of blood cysts or of arachnoid cysts either beneath the seat of injury or on the opposite side of the brain. A mere fissured fracture may result from a blow on the vault, but this is the only common variety of vault fracture which is not accompanied by a further depression of the internal table. In addition to this I would again remind you that fractures of the internal table may be present without the existence of a fracture of the external table, and also that when both tables are involved the internal table is always the more damaged of the two, a very slight depression of the external table almost invariably indicating a far more extensive depression of the internal table. I advocate, therefore, early trephining and exploration in all doubtful cases. The actual danger of the operation is very slight, and though some years ago the mortality after trephining was said to be about 4 per cent., yet the death-rate at the present day is certainly much less than that.

The treatment of comminuted vault fractures.—The important question arises as to whether the comminuted fragments are to be replaced or not. A thorough exploration will generally necessitate the removal of all comminuted fragments, large and small, and the reposition of these fragments, either unaltered or converted into smaller portions, is open to two main objections. First, they act as a source of irritation and consequently increase the tendency to the formation of adhesions

between the dura mater and the bone; and secondly, the actual utility of such methods, with regard to the future union and formation of new bone, is open to grave doubt. The smaller fragments, in any case, are generally absorbed. A third serious objection in compound comminuted fractures is that suppuration not infrequently occurs, the fragments undergoing caries or necrosis, keeping up the irritation and correspondingly increasing the chances of septic meningeal infection. It is advisable therefore to remove all comminuted fragments both in simple and in compound cases and to protect the brain by metal plates cut to a suitable size and shape and pegged or nailed in position. Both gold and aluminium plates are too stiff for moulding to the skull surface, and though pure silver possesses certain advantages over the other two metals yet further research is needed as to the relative advantages of these and other metals. By means of these plates the formation of adhesions between the brain or the dura mater and the scalp is prevented, such adhesions being a common cause of later epilepsy, insanity, &c. The dura mater when torn must be sewn up. If this is impossible all dural tags must be cut away and the future formation of adhesions between the brain and the dura mater and between the dura mater and the bony margins of the gap in the calvarium may be best prevented by the insinuation of thin gold foil between the brain and the dura mater and between the dura mater and the bone. The use of gold foil and of metal plates reduces to a minimum the possible formation of adhesions between any of the structures mentioned. When there is brain laceration, when the subarachnoid space is opened up, and when the chances of suppuration are great the metal plate should then only incompletely close up the bony gap, a small area being left uncovered at the most dependent part to allow of free drainage.

Basic fractures with compression and basic fractures without compression but pointing to a fatal termination.—In these cases death in all probability results from the increased intracranial pressure acting most forcibly on the nuclei in the region of the fourth ventricle. The amount of fluid in the cerebellar fossæ is almost always much increased and I consequently put forward as a suggestion the following mode of treatment. When there is no obvious compressing agent present, such as a middle meningeal hæmorrhage, foreign body, &c., the cerebellar fossæ should be trephined as low down as the muscular attachments will allow, the left fossa being chosen if the injury was inflicted on the anterior part of the right side of the head, whilst if the blow was delivered on the right occipital region the cerebellar

fossa of the same side should be explored. The opening should be of good size, the dura mater opened and the bone not replaced. A drainage-tube should lead through the flap to the gap in the skull. If the patient recovers the gap can, if necessary, be subsequently closed in. The cases for which this treatment is advocated almost invariably have a fatal termination, and the adoption of this method of treatment may add to the chances of recovery since the lessening of the pressure in the region of the fourth ventricle will aid the nuclei to tide over the danger period. In conclusion, I would earnestly plead that all head cases should be treated for a more prolonged period of time, either in the hospital or in the convalescent home. Six months should elapse before the patient goes back to his normal occupation, and as much of this time as is possible should be spent under medical supervision. Iodides and mercury are of some use in hastening the resolution of blood and of inflammatory exudation and in diminishing the general cerebral irritability.

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